PREVALENCE OF NONALCOHOLIC FATTY LIVER DISEASE AND METABOLIC SYNDROME IN PSORIASIS IN A TERTIARY HEALTH CENTER

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CHENNAI

APRIL 2015

CERTIFICATE

This is to certify that the dissertation titled **"PREVALENCE OF NONALCOHOLIC FATTY LIVER DISEASE AND METABOLIC SYNDROME IN PSORIASIS IN A TERTIARY HEALTH CENTER"** is the bonafide original work of in partial fulfillment of the requirements for M.D. Branch-I (General Medicine) Examination of the Tamilnadu DR. M.G.R Medical University to be held in APRIL 2015. The Period of study was from April 2014 to September 2014.

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DECLARATION

I, **Dr.S.ABARNA DEVI** solemnly declare that dissertation titled **"PREVALENCE OF NONALCOHOLIC FATTY LIVER DISEASE AND METABOLIC SYNDROME IN PSORIASIS IN A TERTIARY HEALTH CENTER"** is a bonafide work done by me at Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai-3 during April 2014 to September 2014 under the guidance and supervision of my unit chief Professor of Medicine, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai.

This dissertation is submitted to Tamilnadu Dr. M.G.R Medical University, towards partial fulfillment of requirement for the award of **M.D. Degree (Branch – I) in General Medicine – APRIL 2015.**

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CONTENTS

ABSTRACT

PREVALENCE OF NONALCOHOLIC FATTY LIVER DISEASE AND METABOLIC SYNDROME IN PSORIASIS IN A TERTIARY HEALTH CENTER

Abarna Devi S. R Penchalaiah. K. Narayanaswamy.

INTRODUCTION: Psoriasis vulgaris is a common dermatological condition, worldwide, with a higher prevalence of 0.8-5.6% India. Psoriasis is not just skin deep, and psoriasis patients suffer with many systemic illness directly or indirectly.

AIMS AND OBJECTIVES: To study the prevalence of nonalcoholic fatty liver disease and metabolic syndrome in psoriasis in south Indian population and to correlate the severity of psoriasis with the prevalence of NAFLD .

MATERIALS AND METHODS: Ours is a cross sectional study 6 months, with 165 psoriasis patients. Psoriasis patients on hepatotoxic drugs, significant alcohol use are excluded from the study. After clinical history and examination all the patients are subjected to battery of investigations including ultrasonography.

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RESULTS: The prevalence of NAFLD and metabolic syndrome among psoriasis in our study is 75(45.5%) and 27(16%) respectively. All other components of metabolic syndrome are also increased in our study. There is no association of NAFLD with severity of psoriasis. Patients with psoriasis and metabolic syndrome had significant dyslipidemia.

CONCLUSION: There is a significant association between psoriasis and metabolic syndrome and NAFLD. Psoriasis is independently associated with NAFLD.

KEY WORDS: Psoriasis, NAFLD, metabolic syndrome.

INTRODUCTION

Psoriasis vulgaris as the name implies is a common dermatological condition, worldwide with a prevalence of 1.5%-3% [(1)].A study in India quotes a higher prevalence of 0.8-5.6% ,[(2)] as environmental factors play a role with countries at greater latitudes from equator have a higher prevalence.

It is now recognized that psoriasis is not just skin deep, and psoriasis patients suffer with many systemic illness directly or indirectly .Various studies across the world have demonstrated a chronic systemic inflammatory state of psoriasis which predisposes these patients to a higher relative risk of several comorbidities affecting almost all the system of the body .it is well known that psoriasis patients have a higher prevalence of coronary artery disease[(3)] and suffer early mortality.

Onesuch comorbidity which has gained importance is metabolic syndromeand its sequel which is alarming.

While treating a psoriasis patient, the treating Physician or Dermatologist should keep in mind, regarding various comorbidities associated with psoriasis for the following reasons:

(I)To create an awareness among the psoriasis patients about the various comorbidities

(ii) To educate these patients on various primary and secondary prevention modalities of non-communicable illness which are relatively more prevalent among psoriasis patients.

(iii) Physicians should be aware of the guidelines recommended for monitoring psoriasis patient, for early detection of comorbidities and treatment.

(iv) Physician’s should be aware of the various treatment modalities of psoriasis, to keep a check on drug interactions, while treating systemic illness.

This study is designed to know the magnitude of metabolic syndrome among psoriasis patients in our region .

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES

PRIMARY OBJECTIVES

To study the prevalence of nonalcoholic fatty liver disease in psoriasis in south Indian population.

1. To study the prevalence of metabolic syndrome in psoriasis in south Indian population
2. To correlate the severity of psoriasis with the prevalence of NAFLD
3. To find the association of nonalcoholic fatty liver disease and metabolic syndrome with age, severity and type of psoriasis.

SECONDARY OBJECTIVES

4. To find the association of nonalcoholic fatty liver disease and metabolic syndrome with age, severity and type of psoriasis.
5. To establish psoriasis as a systemic disease and the need for screening of psoriasis patients for NAFLD and metabolic syndrome.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

PSORIASIS VULGARIS

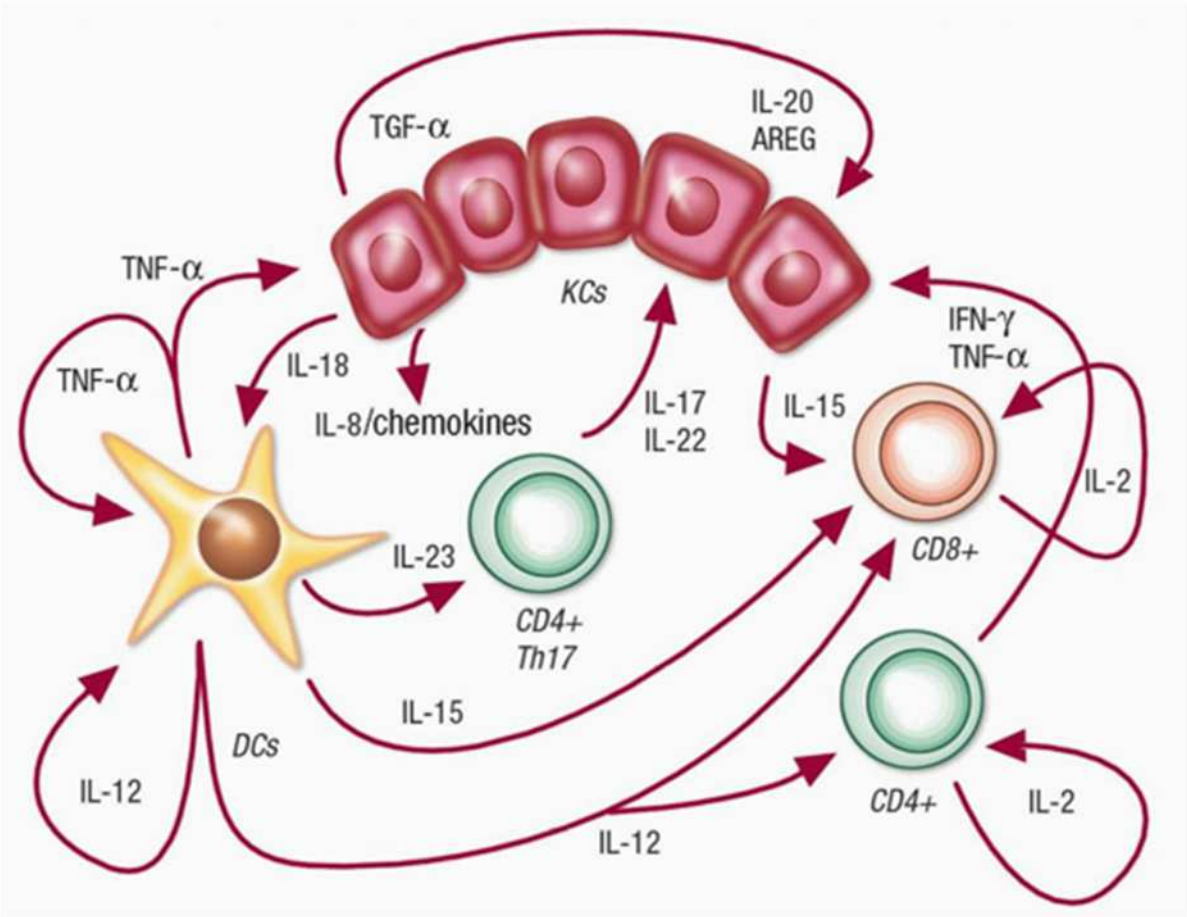
Psoriasis is a multisystemdisease, predominantly affecting skin, nails, and joints. The prevalence of psoriasis is around 1.5 -3% worldwide and it affects both the sex equally, with bimodal distribution. There are two types of psoriasis, type I which is familial, occurs in young age group, with preceding streptococcal infection and type II occurs without any preceding infectious trigger[(1),(13)].

HISTORICAL ASPECTS

“Psora” in Greek means itch .Its first description dates back to biblical times, nearly 3000 years back, as it is documented in Old Testament by Moses .The term psoriasis was first used by Galen.[(4)]

Psoriasis is a multifactorial,multisystem disease, resulting from interplay of genetic and environmental factors, causing dysregulation of innate and adaptive immune system.

PATHOGENESIS



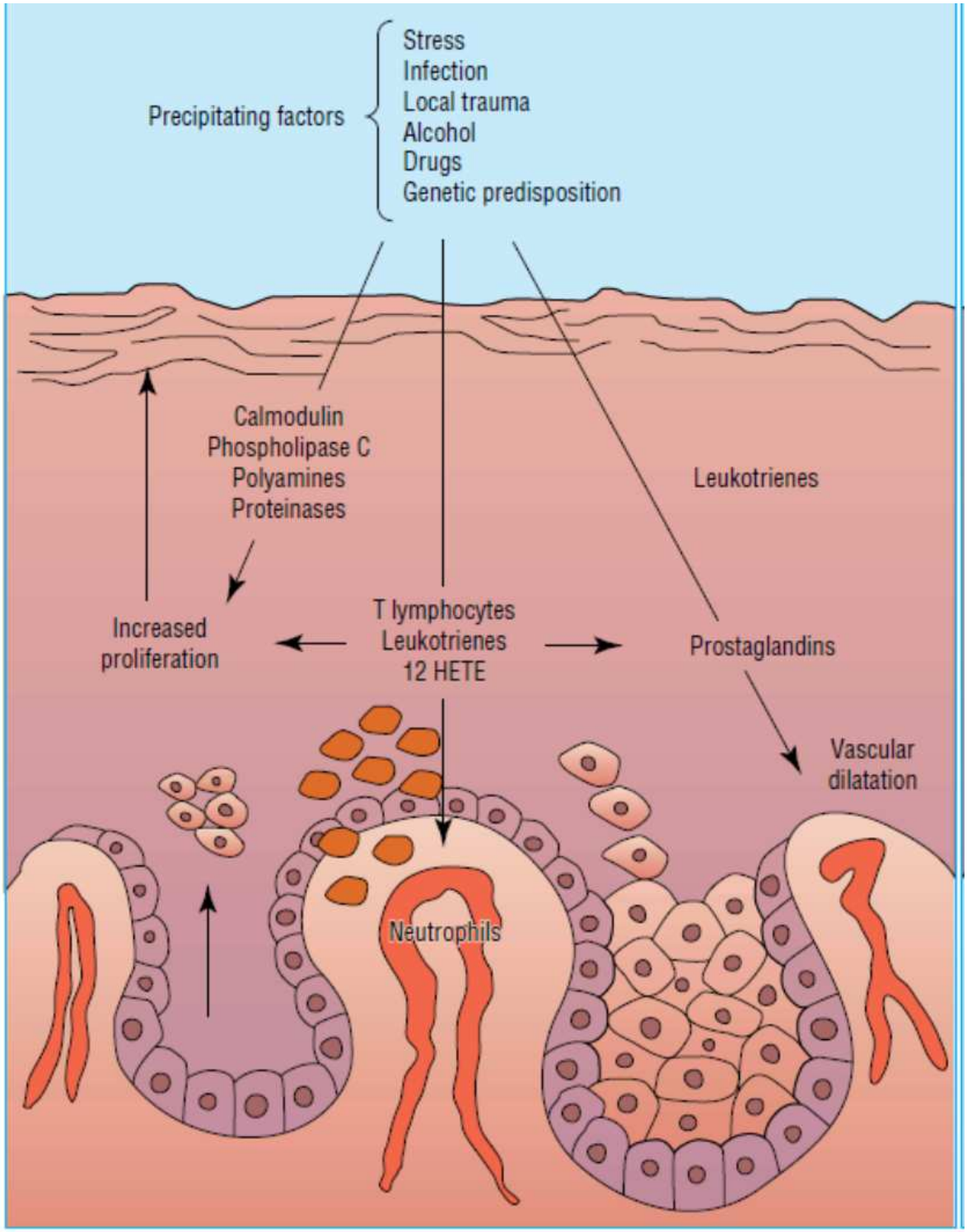
Psoriasis is chronic immune mediated inflammatory state resulting in state of hyper proliferation, predominately mediated by T helper cells. An early overt response of innate immune system to an antigenic trigger is responsible for the immune dysregulation in psoriasis. The recent concept is that psoriasis is mediated by Th17 cells. Stimulation of the antigen presenting cells in epidermis by cytokines like TNF-α, IL-6, IL-1β IFN-α, IFN-γ, due to an unknown antigen results in antigen presentation and release of IL-12 and IL-23 by dendritic cells.[5]

IL-23 is the key cytokine which results in differentiation of T cells to Th17 cells, which in turn release cytokines IL-17 and IL-22 leading to keratinocyte proliferation, production of antimicrobial peptides, proinflammatory cytokines and chemokine[6].

Epidermal proliferation due to keratinocytes mainly occurs in basal and suprabasal layer, predominantly due to TGF alpha cytokine.

ANGIOGENESIS

Psoriasis is characterized by increased angiogenesis[1]. Psoriatic lesions overexpress VEGF which is responsible for the angiogenic activity producing dilated, tall tortuous dermal capillaries. It is also considered responsible for systemic capillary leak in psoriatic patients which manifest as proteinuria, the level of which correlates with serum VEGF. There is up regulation of leukocyte homing molecules such as E-selectin, and ICAM expression on the surface of dermal capillaries due to inflammatory mediators like histamine, neuropeptides, IL-1, TNF alpha, the mechanism behind accumulation of skin homing T- lymphocytes in lesion.


Precipitating factors
Stress
Infection
Local trauma
Alcohol
Drugs
Genetic predisposition
Calmodulin
Phospholipase C
Polyamines
Proteinases
Leukotrienes
Increased proliferation
T lymphocytes
Leukotrienes
12 HETE
Prostaglandins
Vascular dilatation
Neutrophils

GENETICS OF PSORIASIS

Nine chromosomal loci PSORS1-PSORS 9[6] are identified,that are linked to psoriasis.PSORS1 is the major genetic determinant, responsible for nearly 30-50% of familial psoriasis. PSORS 8 gene overlaps with Crohn's disease loci in chromosome 16q,which explains increased occurrence of Crohn's in psoriasis.

ENVIRONMENTAL FACTOR

Several factors could trigger or exacerbate psoriasis like infections with staphylococcus aureus, candida species, malassezia furfur, streptococcus species,as well as smoking, sunlight, trauma are known to exacerbate psoriasis.

DRUGS

Lithium, antimalarial, ACE inhibitors,NSAID'S,beta blockers, clonidine, terfenadine, gemfibrosil, trazadone, potassium iodide, digoxin, amiodarone, penicillin, withdrawal of systemic steroids are known to exacerbate psoriasis.[8]

Other factors which may precipitate or exacerbate psoriasis include, hypocalcaemia, dialysis, alcohol intake, stress, HIV may exacerbate psoriasis

CLINICAL FEATURES OF PSORIASIS

The disease is characterized by recurrent exacerbations and remissions, of itchywell-defined erythematous, thickened, silvery white scaly papules and plaques predominantly affecting the extensor aspect of the body, involving minimal to entire body surface area involvement.

Pustular psoriasis is characterized by the presence of macroscopic pustules and guttate psoriasis is identified by the presence of numerous tiny widespread erythematous scaly papules and plaques. Nails may be involved in 10-80% [(9)]of psoriasis patients affecting both nail bed and nail matrix and is characterized by subungual hyperkeratosis, splinter hemorrhages, oil drop sign, onycholysis. Psoriatic arthritis affects 10-30% of psoriasis patients.

DIAGNOSIS

Psoriasis is mainly clinical and histopathology of the skin lesion is confirmatory. Psoriatic lesion may exhibit Auspitz sign[(10)],which is removal of thinned epidermis by scrapping, reveals multiple vascular bleeding points.

HISTOPATHOLOGY

Psoriatic lesion reveals parakeratosis, orthokeratosis, munromicro abscess, thinning of granular layer, elongation and thinning of rete ridges, suprapapillarythinning of epidermis.[11]

MORPHOLOGICAL TYPES OF PSORIASIS

1. Chronic plaque psoriasis

2. Pustular psoriasis[12]

3. Erythrodermic psoriasis

4.Guttate psoriasis

5. Psoriatic arthritis

6. Mucous membrane psoriasis

7. Rupioid , Elephantine and Ostraceous psoriasis.

8. Follicular psoriasis

9. Psoriasis unguis/nail psoriasis

11. Unstable psoriasis

12. Linear and Zonal forms[14]

13. Sebopsoriasis

SEVERITY ASSESSMENT SCORES FOR PSORIASIS

1) Psoriasis area and severity index (PASI)

2) Dermatologylife quality index (DLQI)

3) Body surface area (BSA)

4) Visual analogue scale (VAS)

5) Short form -36

CALCULATION OF PSORIASIS AREA AND SEVERITY INDEX (PASI) :

PASI score ranges from 0 to 72.it is the most widely used objected method to assess the disease severity and response to treatment

CALCULATION OF THE PSORIASIS AREA AND SEVERITY INDEX (PASI)				
Severity of psoriatic lesions [0, none; 1, slight; 2, moderate; 3, severe; 4, very severe]				
	Head	**Trunk**	**Upper limbs**	**Lower limbs**
Erythema	0 to 4	0 to 4	0 to 4	0 to 4
Induration	0 to 4	0 to 4	0 to 4	0 to 4
Scaling	0 to 4	0 to 4	0 to 4	0 to 4
Total score = **1**	Sum of the above	Sum of the above	Sum of the above	Sum of the above
Area of psoriatic involvement [0, none; 1, <10%; 2, 10 to <30%; 3, 30 to <50%; 4, 50 to <70%; 5, 70 to <90%; 6, 90–100%]				
Degree of involvement = **2**	0 to 6	0 to 6	0 to 6	0 to 6
Multiply **1 × 2**	**1 × 2**	**1 × 2**	**1 × 2**	**1 × 2**
Correction factor for area of involvement = **3**	0.10	0.30	0.20	0.40
1 × 2 × 3	**A**	**B**	**C**	**D**
A + B + C + D = total PASI				

PSORIASIS –A SYSTEMIC ILLNESS

Various studies across the world have proved that psoriasis is associated with many other systemic conditions. Multiple organ involvement is known to occur in psoriasis. Hence it is proved beyond doubt that psoriasis is a systemic illness[15]. The comorbidities of psoriasis may be described based on the system affected.

MUSCULOSKELETAL SYSTEM

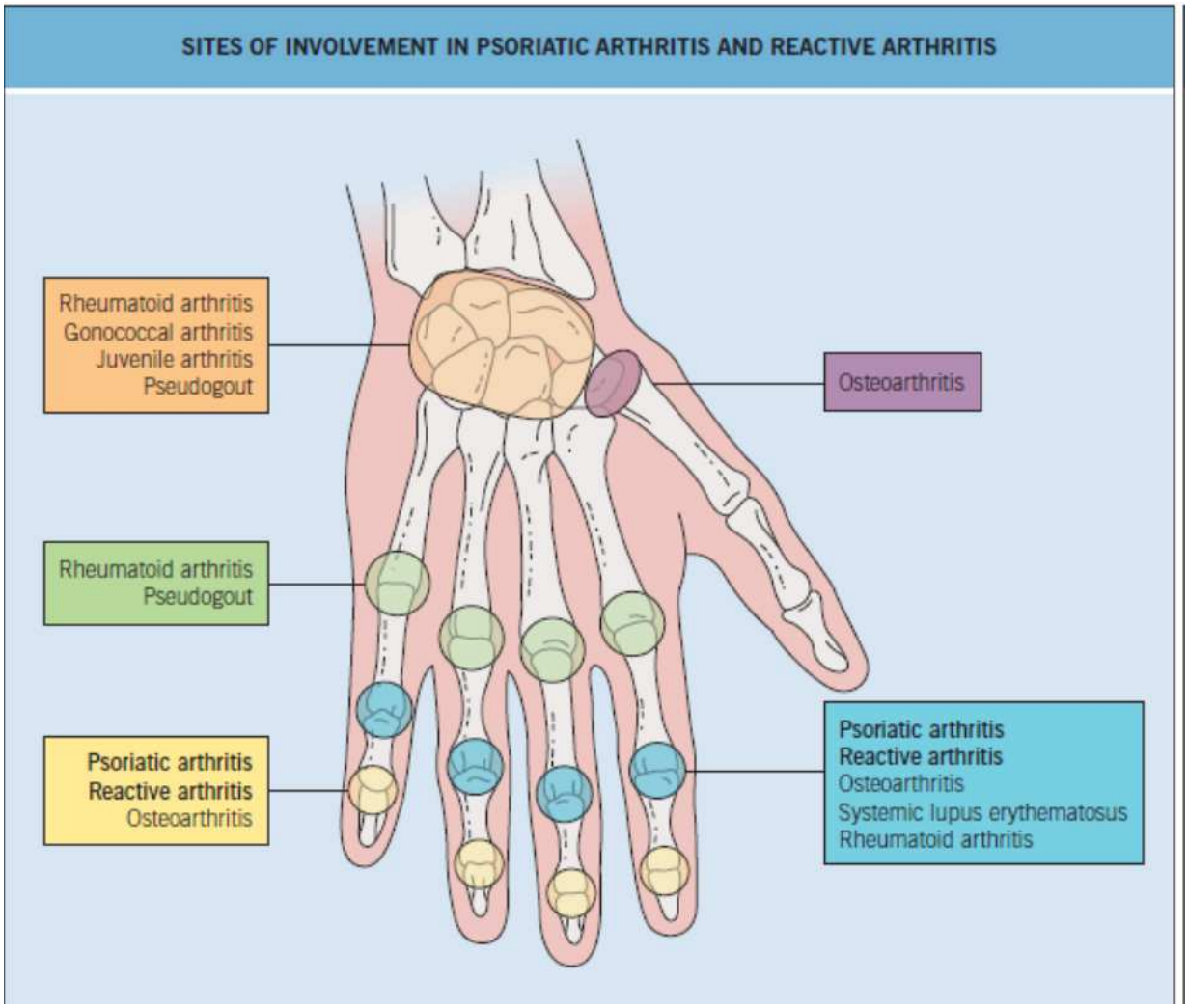
Psoriatic arthritis, osteoporosis, gout, chronic recurrent multifocal osteomyelitis can occur with pustular psoriasis, SAPHO syndrome[16]

PSORIATIC ARTHRITIS:

It is a chronic inflammatory arthropathy involving peripheral joints, spine, and entheses.It manifests as pain, swelling, tenderness of joints, tendon, and ligaments.It is considered as seronegative arthritis, in the sense it is negative for both rheumatoid factor and anti CCP[18]. It may precede,accompany or follow psoriatic skin lesion. PsA may progress to erosive arthritis with joint destruction .the prevalence of PsA is more in severe psoriasis and prevalence of nail psoriasis is more in patients with PsA .An association with HLA B 27 is well known with PsA.[17]

MOLL AND WRIGHT CLASSIFICATION OF PSORIATIC ARTHRITIS:

Type	Percentage of all psoriatic arthritis	Features
Oligoarticular, asymmetric arthritis (one or more joints)	30-50	Joints of fingers and toes ("sausage finger") are involved.
Polyarticular, symmetric arthritis (RA-like)	30-50	Clinically resembles rheumatoid arthritis, rheumatoid factor negative. The small joints of the hands and feet, wrists, ankles, knees, and elbows may be involved.
Distal interphalangeal joint predominant	25	Mild, chronic, not disabling, and associated with nail disease. Involves hands and feet. This is the most characteristic presentation of arthritis with psoriasis.
Destructive polyarthritis (arthritis mutilans)	5	The most severe form of psoriatic arthritis involves osteolysis of any of the small bones of the hands and feet. Gross deformity and subluxation are attributed to this condition. Severe osteolysis leads to digital telescoping, producing the "opera glass" deformity. This deformity may be seen in rheumatoid arthritis.
Ankylosing spondylitis and sacroiliitis	30-35	Occurs as an isolated phenomenon or in association with peripheral joint disease. Association of HLA-B27 and spondylitis. The strongest association is in males with sacroiliitis. Asymptomatic sacroiliitis occurs in as many as one third of cases of psoriasis. It is usually asymmetric and may be associated with spondylitis.


SITES OF INVOLVEMENT IN PSORIATIC ARTHRITIS AND REACTIVE ARTHRITIS
Rheumatoid arthritis
Gonococcal arthritis
Juvenile arthritis
Pseudogout
Osteoarthritis
Rheumatoid arthritis
Pseudogout
Psoriatic arthritis
Reactive arthritis
Osteoarthritis
Psoriatic arthritis
Reactive arthritis
Osteoarthritis
Systemic lupus erythematosus
Rheumatoid arthritis

Differentiating Psoriatic Arthritis from Rheumatoid Arthritis

	Psoriatic arthritis	Rheumatoid arthritis	Inflammatory osteoarthritis	Gout
Distal interphalangeal joint involvement	Common	Uncommon	Common	Uncommon
Symmetry	Less common	Common	Uncommon	Uncommon
Erythema of joint	Common	Uncommon	Uncommon	Common
Stiffness	In morning and/or with immobility	In morning and/or with immobility	With activity	Uncommon
Tenderness	Mild	Severe	Mild	Severe
Back involvement	Common	Uncommon	Uncommon	Uncommon
Skin lesions	Always	Uncommon	Uncommon	Uncommon
Nail lesions	Common	Uncommon	Uncommon	Uncommon
Dactylitis	Common	Uncommon	Uncommon	Uncommon
Enthesitis	Common	Uncommon	Uncommon	Uncommon
Rheumatoid nodules	Never	Common	Uncommon	Uncommon
Rheumatoid factor	Uncommon	Common	Uncommon	Uncommon
Radiological changes				
Osteopenia	Less common	Common	Uncommon	Uncommon
Periostitis	Common	Rare	Uncommon	Uncommon
Pencil-in-cup change	Common	Rare	Uncommon	Uncommon
Ankylosis	Common	Rare	Uncommon	Uncommon
Sacroiliitis	50%, asymmetric	Rare	Uncommon	Uncommon
Quality of life	Reduced	Reduced	Reduced	?
Function	Reduced	Reduced	Reduced	?
HLA association	CW6, B27	DR4	No	B14
Female to male ratio	1:1	3:1	Hand/foot more common in female patients	1:3.6

DIFFERENTIATING PSORIASIS FROM SPONDYLOARTHROPATHIES

Feature	Psoriatic arthritis	Ankylosing spondylitis	Reactive arthritis	Inflammatory bowel disease
Male to female ratio	1:1	3:1	8:1	1:1
Age at onset (years)	35-45	20	20	Any
Peripheral distribution	96% Any	25% Axial Lower limbs	90% Lower limbs	Common Lower limbs
Dactylitis	35%	Uncommon	Common	Uncommon
Enthesitis	Common	Common	Common	Uncommon
Sacroiliitis	50%	100%	80%	20%
Syndesmophytes	Classic and paramarginal	Classic	Classic and paramarginal	Classic
HLA-B	50%	>90%	80%	40%
Psoriasis	Always	Uncommon	Uncommon	Uncommon
Other skin lesions	Nail changes	Uncommon	Keratodermia blennorrhagica	Erythema nodosum, pyoderma gangrenosum
Quality of life	Reduced	Reduced	Likely reduced	Reduced
Function	Reduced	Reduced	Reduced	Reduced

GASTROINTESTINAL SYSTEM: [19]

Crohnsdisease, ulcerative colitis, celiac disease, gastro esophageal reflux disease is more prevalent among psoriasis patients.

PSORIASIS AND LIVER

Psoriasis patents have higher relative risk of developing nonalcoholic fatty liver disease (NAFLD) and viral hepatitis .psoriasis patients suffer from drug induced hepatotoxicity due to methotrexate, acitretin, etc.[20]

The prevalence of NAFLD among psoriasis is around 48-59%. Correlation between NAFLD and severity of psoriasis is not well established.

Gisondi et al[21] study quotes that NAFLD was associated with the severity of psoriasis. Whereas Miele et al study showed NAFLD to be unrelated to psoriasis severity.Other finding from the same study is that psoriatic patients with NAFLD were more likely to have psoriatic arthritis.

RENAL SYSTEM

Case reports of acute tubular necrosis, poststreptoccocal glomerulonephritis have been described following pustular and guttate psoriasis respectively. Renal failure due to amyloidosis may be a sequel to arthropathic, pustular and severe non pustular psoriasis.[22]

RESPIRATORY SYSTEM

Chronic obstructive pulmonary disease(COPD) is more common in psoriasis. Apical pulmonary fibrosis is reported to occur in association with psoriatic arthritis.[23]

OCULAR SYSTEM

Uveitis can occur in pustular psoriasis and psoriatic arthritis. Other ocular complications includes keratitis, blepharitis, conjunctivitis, symbhlepharon, xerosis.

MALIGNANCY

Psoriatic patients are at increases risk of developing non melanoma skin cancer and lymph proliferative malignancy probably due to immunotherapeutic agents and phototherapy.

Lip, oral, pharyngeal, peritoneum carcinoma are also more common among psoriasis.

CENTRAL NERVOUS SYSTEM

Psoriasis is a pre atherosclerotic state,cerebrovascular accident are more common among psoriasis patients. It is one of the causes for mortality in psoriasis patients.

Psoriasis more commonly occurs in families with multiple sclerosis.

PSYCHOSOCIAL ASPECTS OF PSORIASIS

Depression in psoriasis may be as high as 60%.They may also suffer from low self-esteem, sexual dysfunction,anxiety and sleep disorder

Smoking and alcohol consumption is more common among psoriasis patients compared to the general population.

CADIOVASCULAR SYSTEM

Epidemiological studies have found an association between psoriasis and cardiovascular risk factors such as obesity, hypertension, dyslipidemia, diabetes mellitus, and the so called “Metabolic syndrome” the deadly quartet with psoriasis. Psoriasis is considered a state of preatherosclerolsis and hence is an independent risk factor for myocardial infarction conferring early mortality among psoriasis patients. [30]

Presence of hyperuricemia, phototherapy one of the treatment modalities of psoriasis contributes to atherosclerosis risk among psoriasis. Early use of methotrexate for psoriasis significantly reduced the atherosclerosis risk among psoriasis. [28]

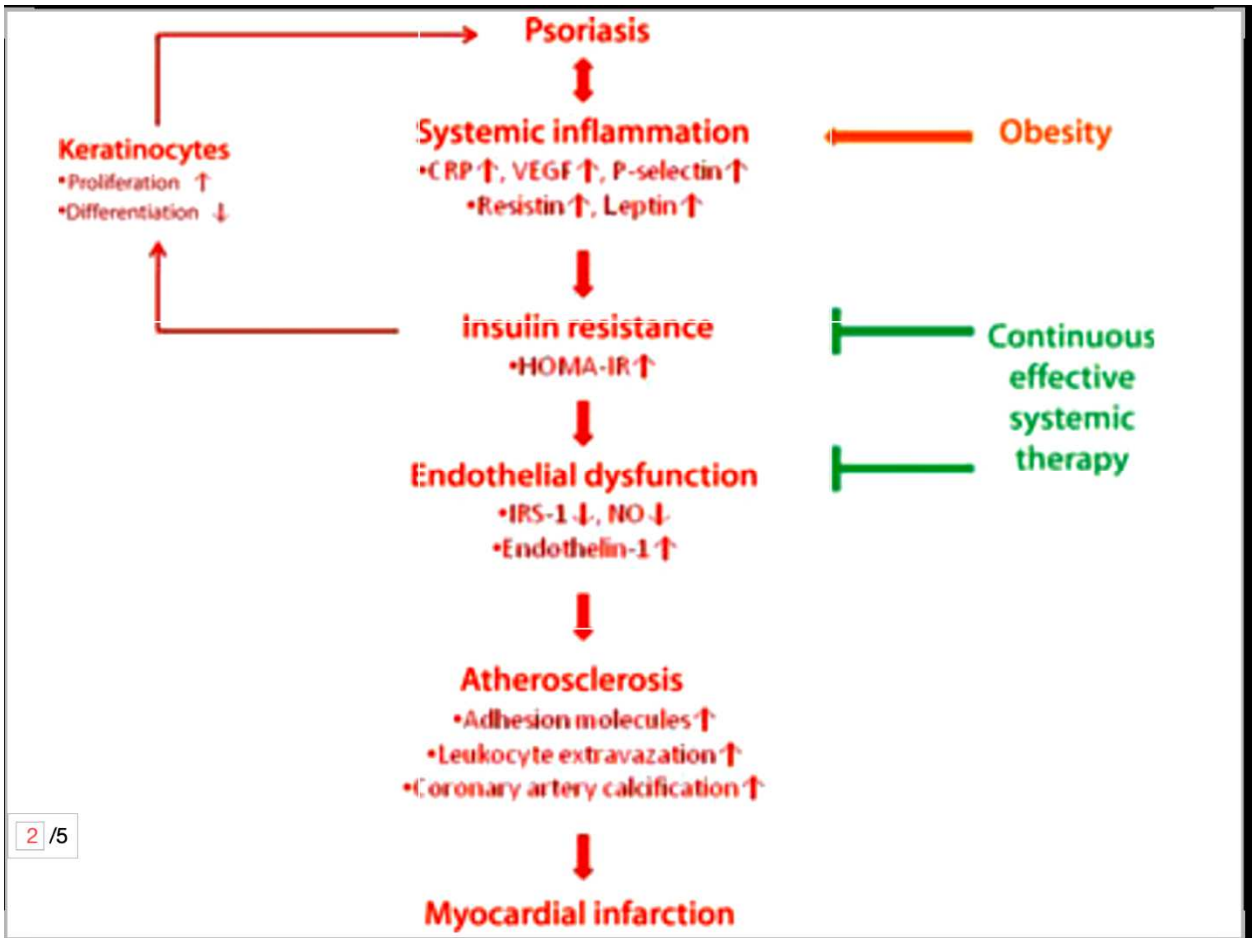
PSORIASIS AND METABOLIC SYNDROME

Concept of psoriatic march:

Chronic systemic inflammation causes a state of insulin resistance, which triggers endothelial cell dysfunction, which leads to atherosclerosis, resulting in CVD, CAD [29]

Continuous effective systemic therapy may stop the psoriatic march.

PSORIATIC MARCH



OBESITY

A study from UK has shown that psoriasis patients are 1.18 times higher risk of developing obesity.A study in Israel comparing obesity in psoriasis patients and control showed a significant p value .In this study obesity was present in 8.4% and 3.6% of psoriasis patients and controls respectively.

Obesity produces a state of a chronic inflammatory state as the immune cells like macrophages infiltrates the adipose tissue, stimulates the production of adipokines.Adipokines are the key cytokines in pathogenesis of adipokines.

Adipokines are the cytokines produced by adipose tissue, the largest endocrine organ.It is shown in studies that adipokines may be used as a biomarker for assessing the occurrence of comorbidities, severity of psoriasis. Various studies across the world have demonstrated that psoriatic patients are at risk of increase in BMI. It is stated that obesity predisposes to psoriasis and vice versa. A study conducted in China cites co-occurrence of obesity and HLA Cw6 in a patient had 35 fold higher risk of developing psoriasis.

PSORIASIS AND ADIPONECTIN

Obesity is associated with decreased adiponectin, as proved in various studies psoriasis patients who are obese had decreased adiponectin level. Proinflammatory cytokines like Il-6 may suppress adiponectin. [31]

PSORIASIS AND LEPTIN

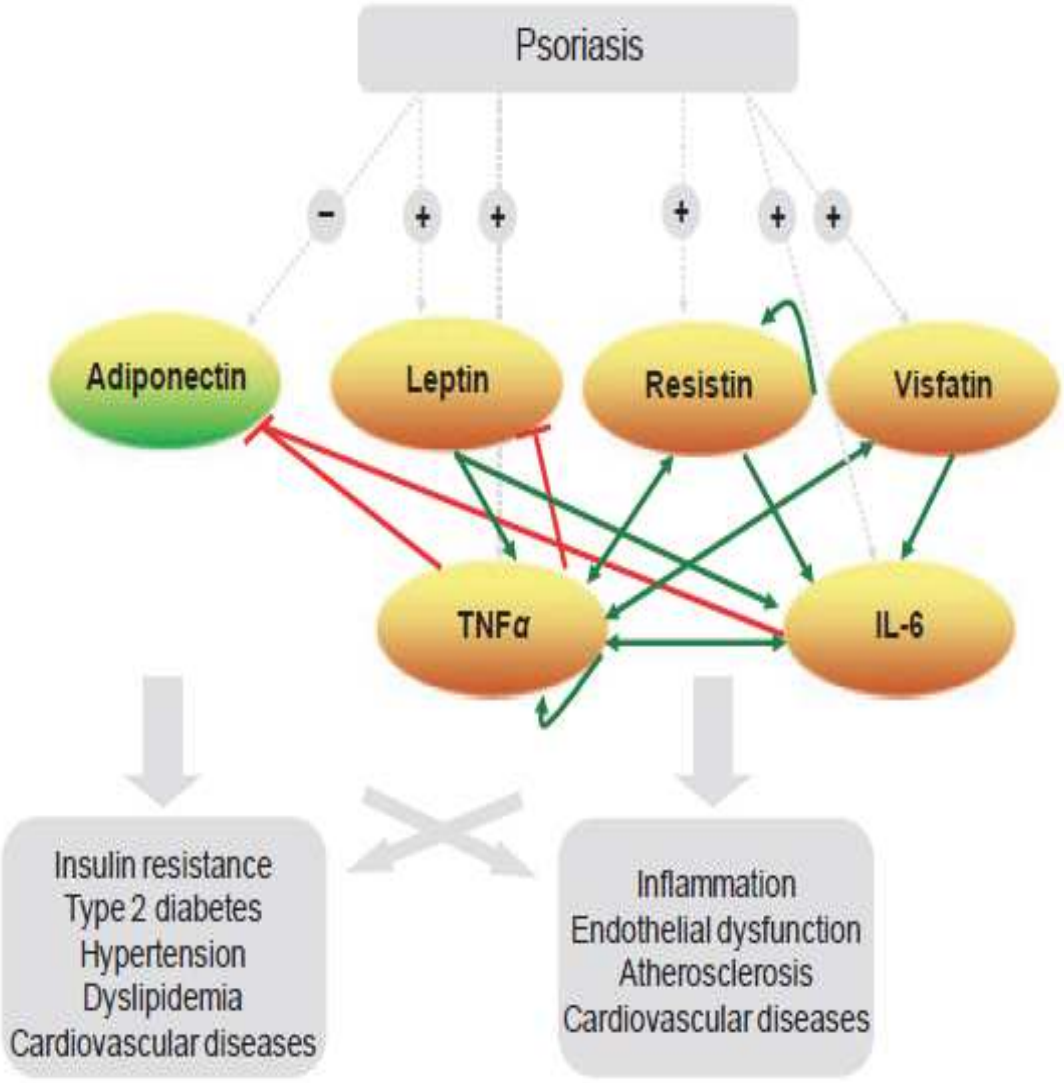
The functions of leptin one of the adipokine is to regulate the appetite, body weight by a feedback mechanism.other action includes skin regeneration, hair growth. Experiments have shown high levels of leptin acts as a proinflammatory mediator, resulting in Th1 cell response.Elevated levels of leptin is associated with intimal media thickness of great vessels and hence it is a marker of coronary artery disease and cardiovascular morbidity.Few reports have shown a positive correlation of leptin levels with the chronicity and duration of psoriasis, and thus it is involved in metabolic syndrome. [32]

PSORIASIS AND RESISTIN

Proinflammatory cytokines like TNF alpha,IL-6, increases the levels of resistin which in turn increases IL-12, TNF alpha ,which is a mediator of endothelial dysfunction and atherosclerosis.Similar to rhematoid arthritis, IBD, psoriasis disease also showed a positive correlation with resistin and its level decreased with systemic treatment of psoriasis.

RBP4 is a marker of systemic insulin resistance and diabetes and found to be increased in psoriasis. omentine increases the sensitivity of insulin and helps in glucose uptake by adipocytes,its level remains unaltered in psoriasis

MAJOR ADIPOKINES IN PSORIASIS:



Green arrow-up regulation:

Red arrows -down regulation

ADIPOKINES IN PSORIASIS:

Table 2. Possible consequences due to altered adipokines in psoriasis

Adipokine/cytokine	Known function(s)	Status in psoriasis	Possible consequences
Adiponectin	Anti inflammatory, anti atherogenic	Down regulated	Development of vascular diseases and metabolic syndrome
Leptin	Control of fat stores, regulation of appetite and body weight; pro inflammatory	Up regulated	Development of vascular diseases
Resistin	Pro inflammatory	Up regulated	Development of vascular diseases
Visfatin	Insulin like effects, pro inflammatory, T cell activation	Up regulated	Development of vascular diseases
RBP4	Transport protein for vitamin A, development of insulin resistance	Not known	If up regulated: Development of metabolic diseases (e.g. insulin resistance, type 2 diabetes)
Omentin	Increase of insulin sensitivity	Not known	If down regulated: Development of metabolic diseases (e.g. insulin resistance, type 2 diabetes)
TNFα	Pro inflammatory	Up regulated	Development of vascular diseases and metabolic syndrome
IL 6	Pro and anti inflammatory	Up regulated	Development of insulin resistance, type 2 diabetes and vascular diseases

COMPARISON OF STUDIES :

PREVALENCE OF METABOLIC SYNDROME AMONG PSORIASIS IN VARIOUS STUDIES

Study	No of pateints	Prevalence of MS	Results	Prevalence of components of MS	Correlation with severity of psoriasis
Safiye kutlu et al	250	30.8	Type 2 psoriasis was more common		
Gisondi et al	338	30.1	MS was common in 1.35.2% of PA 2.>40 years	No significant difference in prevalence on low HDL, FPG, hypertension	1.No correlation of MS 2.Correlates withTGL
Sommer et al			MS more in Moderate –severe psoriasis		
Takahashi et al					No correlation
Nazhatun nisa et al	150	28		Higher prevalence of TGL, FPG, HT	

PREVALENCE OF DYSLIPIDEMIA AMONG PSORIASIS PATIENTS

study	No.of patients	Dyslipidemia	HDL	TGL	Chol	LDL	VLDL
UK	44,164	4.3% (1.17 times)					
Iran		raised	No change	raised		raised	raised
Zarijavidi et al		raised	No change	raised		raised	raised
Turkey	84	raised		raised	raised	raised	
hyderabad	79	raised	No change	raised	raised	raised	No change
Iran	30	No change	No change	No change	No change	No change	No change

PREVALENCE OF CORONARY ARTERY DISEASE IN PSORIASIS:

Azfar et al study with 32 patients based on coronary artery calcification showed a higher prevalence of CAD (59%) among severe psoriasis.

A population based cohort study conducted in UK with 1,30,000 psoriasis patients showed that young patients with severe psoriasis have a higher relative risk of 3.1 of developing CAD .

Another study states that psoriasis patients have 1.21 times higher risk of developing CAD. [33]

HYPERTENSION

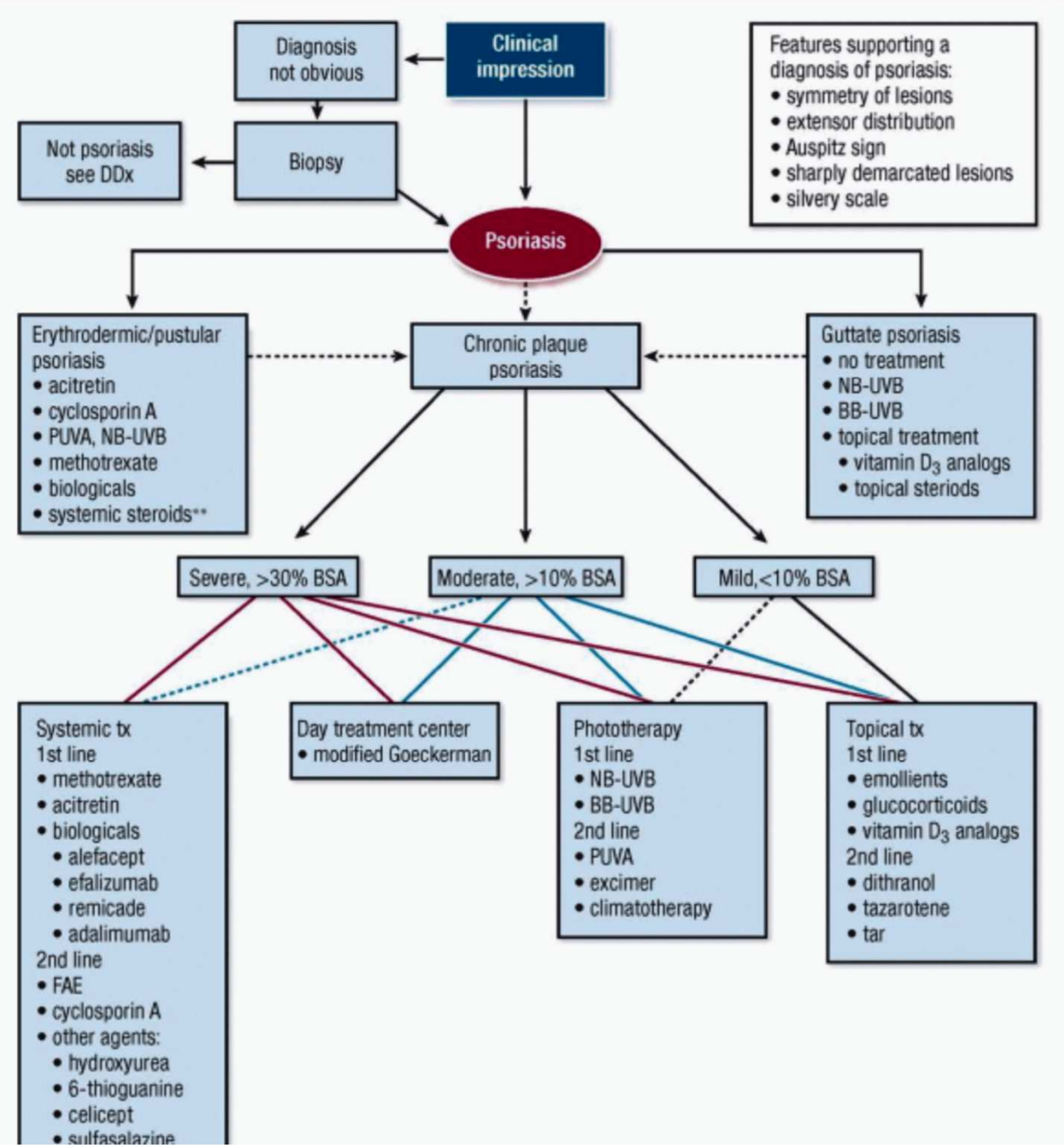
UK studied a prevalence of 6.3% psoriasis patients with hypertension. It is especially common among psoriasis patients with smoking, and alcoholics. Yet another study conducted in Israel states a prevalence of 27.5% of psoriasis patients with hypertension.

GUIDELINES FOR MONITORING PATIENTS FOR METABOLIC SYNDROME

Parameter	Recommendation by the American Heart Association (58)	AJC editor consensus (59)
Blood pressure	• Evaluate at least every 2 years • Target <120/80 mm Hg	• <140/90 mm Hg in all patients with psoriasis and ≤2 major risk factors for CAD • <130/80 mm Hg in patients with previous CVD, diabetes mellitus, chronic renal disease or ≥3 major risk factors
Body mass index	• Evaluate at least every 2 years • Target <25 kg/m^2	Not addressed
Waist circumference	• Evaluate at least every 2 years • Target ○ <102 cm (males) ○ <88 cm (females)	Not addressed
Pulse	Evaluate at least every 2 years	Not addressed
Fasting blood lipids	• Evaluate at least every 5 years or every 2 years if risk factors[1] are present • Total cholesterol ≤ 200 mg/dl • LDL ○ Optimal: <100 mg/dl ○ Near optimal: 100–129 mg/dl ○ Borderline: 130–159 mg/dl ○ High: 160–189 mg/dl ○ Very high: ≥190 mg/dl	• 1 CAD risk factor: LDL < 160 mg/dl • ≥2 CV risk factors: LDL < 130 mg/dl • If CVD present or CAD risk equivalents[2]: LDL < 100 mg/dl
Fasting blood glucose	• Evaluate at least every 5 years or every 2 years if risk factors[1] are present • Target: <100 mg/dl	Not addressed

TREATMENT OF PSORIASIS:

Both topical and systemic therapies are available for the treatment of psoriasis Treatment of psoriasis should be individualized.



TREATMENT OF PSORIASIS WITH BSA < 20%

Treatment	Advantages	Disadvantages
Topical steroids	Rapid response, controls inflammation and itching, best for intertriginous areas and face, convenient, not messy	Temporary relief (tolerance occurs), less effective with continued use, atrophy and telangiectasia occur with continued use, brief remissions, very expensive
Calcipotriol (Dovonex)	Well tolerated, long remissions possible	Burning, skin irritation, expensive
Tazarotene (Tazorac)	Effective, long remissions possible	Irritating, expensive
Anthralin	Convenient short-contact programs, long remissions, effective for scalp	Purple-brown staining, irritating, careful application (only to plaque) required
Tar	New preparations are pleasant	Only moderately effective in a few patients
UVB and lubricating agents or tar	Insurance may cover part or all of treatment, effective for 70% of patients, no need for topical steroids	Expensive, office-based therapy
Tape or occlusive dressing	Convenient, no mess	Expensive, only for limited disease
Intralesional steroids	Convenient, rapidly effective, long remissions	Only for limited areas, atrophy and telangiectasia occur at injection site

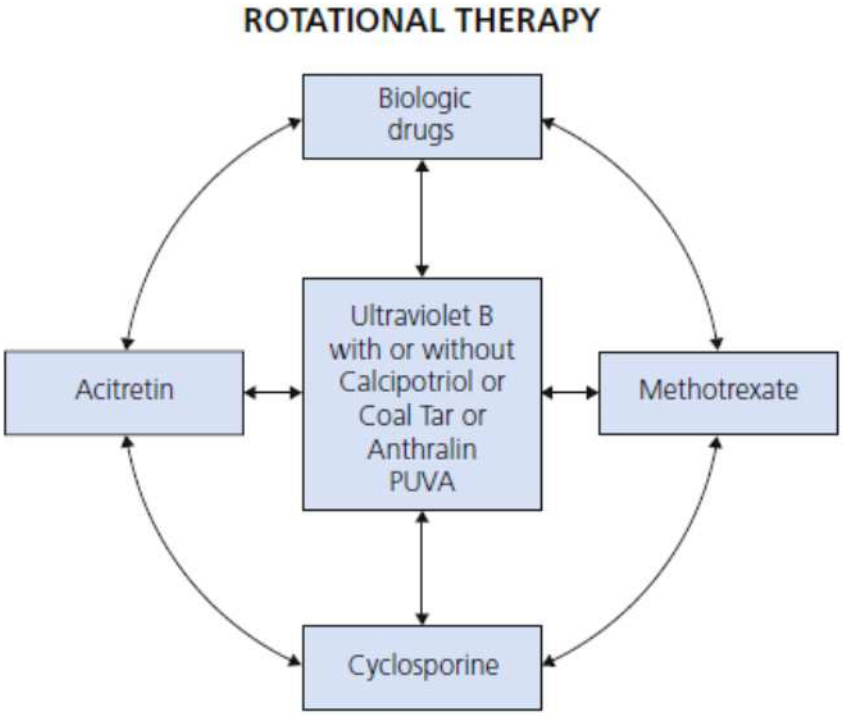
TREATMENT OF PSORIASIS WITH BSA > 20%

Treatment	Advantages	Disadvantages
UVB and narrow-band ultraviolet B light (NB-UVB)	Effective and safe	Requires many office visits
PUVA	Allows patient to be ambulatory; effective	Many treatments needed; many office visits required, carcinogenic
Methotrexate	"Gold standard" for efficacy; helps arthritis	Hepatotoxicity; liver biopsy periodically required
Acitretin (Soriatane)	Effective for palmar-plantar-pustular, erythro-dermic, and pustular types of psoriasis; fast, effective; helps arthritis	Teratogenic; many annoying side effects
Cyclosporine	Fast, effective; helps arthritis	Nephrotoxic; immu-nosuppressive; expensive

BIOLOGICAL THERAPIES IN PSORIASIS

COMMERCIALLY AVAILABLE BIOLOGIC AGENTS U THE TREATMENT OF PSORIASIS		
Biologic agent	**Target**	**Molecule**
Alefacept	CD2	Human fusion protein
Efalizumab	CD11a	Humanized antibody
Etanercept	TNF-α^	Human fusion protein
Infliximab	TNF-α^^	Chimeric antibody
Adalimumab	TNF-α^^	Human antibody

Rotation of available therapy for moderate to severe psoriasis may avoid long term cumulative systemic toxicity and use of drugs effectively and wisely.



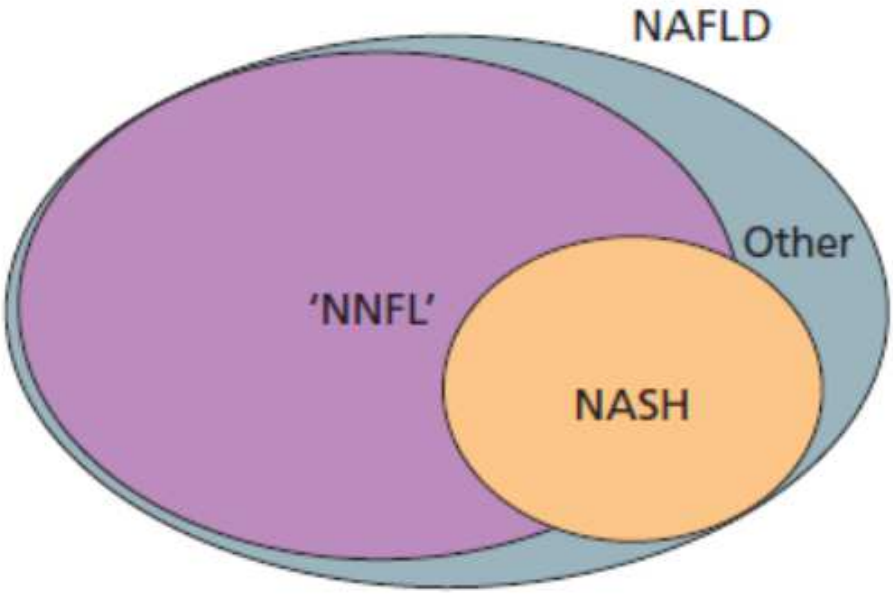
NON ALCOHOLIC FATTY LIVER DISEASE

Cryptogenic cirrhosis accounts for approximately 10-20% of causes for cirrhosis, of which nonalcoholic fatty liver disease (NAFLD) is the most common cause.

It was Morgan in 1870 who initially described an association of fatty liver with obesity. In 1980, Ludwig et al observed characteristic features of alcoholic liver disease in liver biopsy specimens in patients who did not consume alcohol, neither used drugs that causes steatohepatitis. Hence he coined the term nonalcoholic steatohepatitis (NASH).

Nonalcoholic fatty liver disease (NAFLD) is a spectrum of condition ranging in severity from mild liver steatosis to very severe disease NASH which may progress to cirrhosis, liver failure, and hepatocellular cancer in patients without significant ethanol exposure. NAFLD is the most common liver disease in the world. [34]

SPECTRUM OF NAFLD



DEFINITIONS AND TERMINOLOGY

The criteria to diagnose NAFLD in a patient necessitates exclusion of all other possible etiologies which can cause liver steatosis, such as significant alcohol consumption ,viral hepatitis, drug - induced injury ,Wilson ’s disease, hemochromatosis and α 1 - antitrypsin deficiency .

Significant alcohol consumption for causing fatty liver is estimated as 30 g/day in males and 20 g/day in females. [35]

NON ALCOHOLIC FATTY LIVER DISEASE(NAFLD):

It is defined as fatty infiltration of more than 5-10% of liver by weight, or more than 5–10% of hepatocytes in biopsy specimens by light microscopy without any evidence of cell injury.

NONALCOHOLIC STEATOHEPATITIS (NASH):

It is the severe form of NAFLD wherein fatty infiltration of liver is associated with inflammation, ballooned hepatocytes, necroapoptosis , and fibrosis, usually beginning around the central vein.

NON NASH FATTY LIVER (NNFL):

It is synonymous to nonalcoholic fatty liver (NAFL), /simple steatosis, with minimal or no inflammation, and no fibrosis.

PRIMARY NAFLD/NASH:

If NAFLD or NASH is associated with central obesity, diabetes mellitus type 2, without any other specific etiology then it is primary NAFLD.

SECONDARY NAFLD or NASH:

If NAFLD or NASH is due to drug inducedor toxin induced then it is secondary NAFLD. It is also called as toxin associated steatohepatitis(TASH)

.EPIDEMIOLOGY

NAFLD is usually detected in 4^{th} or 5^{th} decade of life .Population based studies have documented NAFLD in 10-24% of the population to as high as 79% among obese individuals. It is more prevalent among Hispanics than other race.

ETIOLOGY OF NAFLD

Metabolic –congenital or acquired, and drugs

METABOLIC CAUSSES:

CONGENITAL:

Inborn Errors of Metabolism
Abetalipoproteinemia
Familial hepatosteatosis
Galactosemia
Glycogen storage disease
Hereditary fructose intolerance
Homocystinuria
Systemic carnitine deficiency
Tyrosinemia
Weber-Christian syndrome
Wilson disease

Acquired Metabolic Disorders
Diabetes mellitus
Dyslipidemia
Kwashiorkor and marasmus
Obesity
Starvation

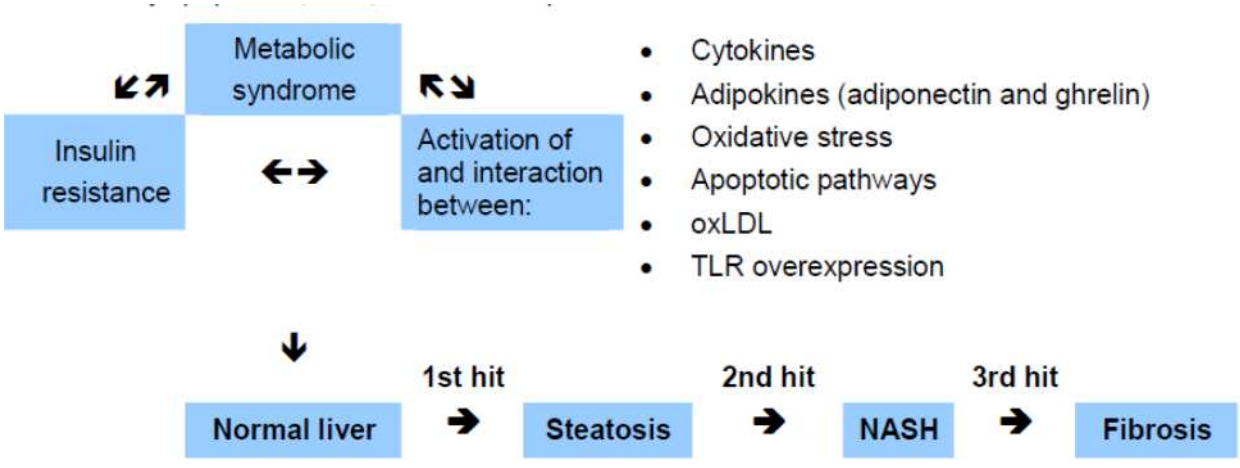
Metals
Antimony
Barium salts
Chromates
Phosphorus
Rare earths of low atomic number
Thallium compounds
Uranium compounds

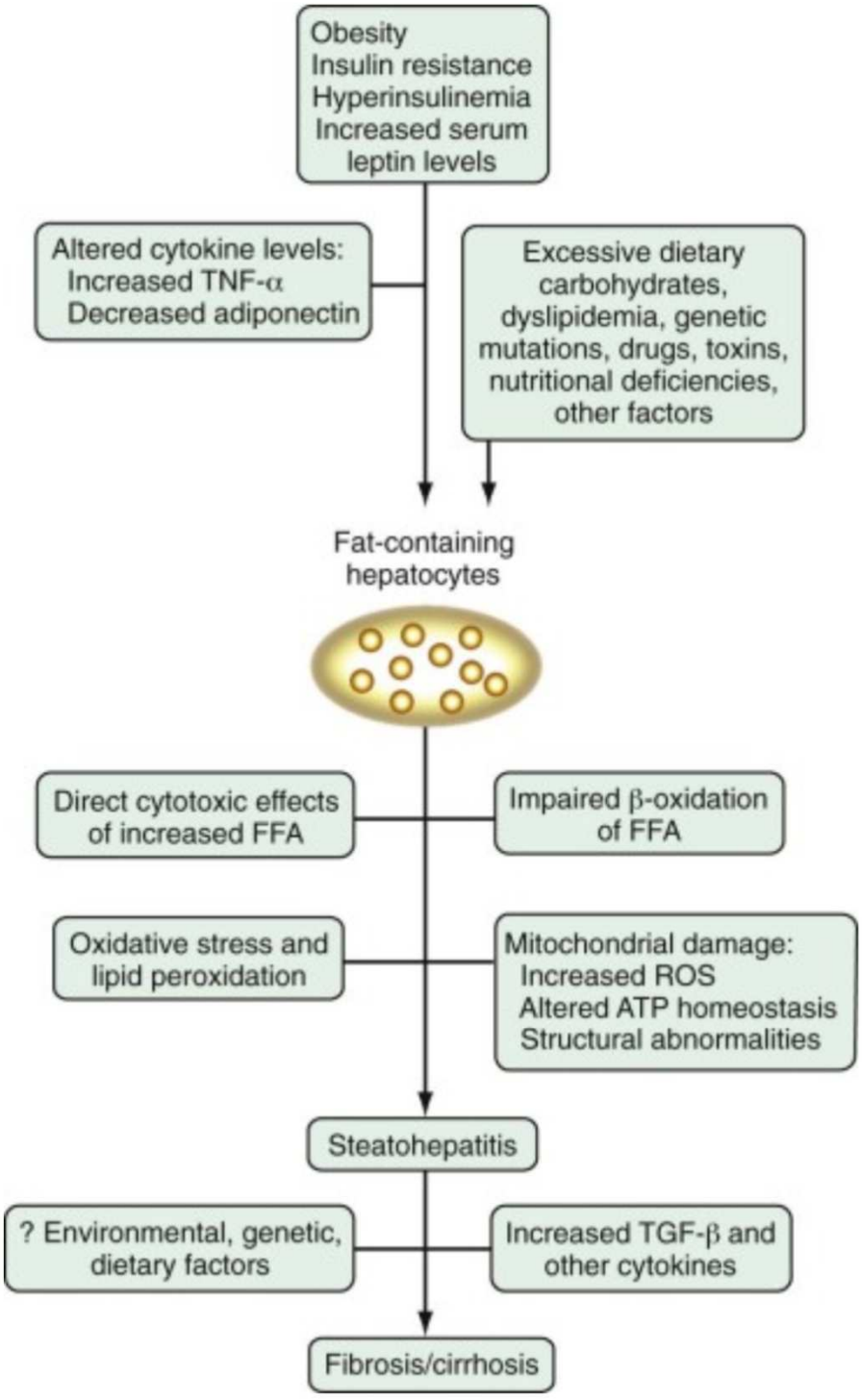
Cytotoxic and Cytostatic Drugs
L-Asparaginase
Azacitidine
Azaserine
Bleomycin
Methotrexate
Puromycin
Tetracycline*
Other Drugs and Toxins
Amiodarone
4,4′-diethylaminoethoxyhexestrol
Dichlorethylene
Ethionine
Ethyl bromide
Estrogens
Glucocorticoids
Highly active antiretroviral therapy
Hydrazine
Hypoglycin
Orotate
Perhexilene maleate
Safrole
Tamoxifen

Surgical Procedures
Biliopancreatic diversion Extensive small bowel resection Gastric bypass Jejunoileal bypass
Miscellaneous Conditions
Industrial exposure to petrochemicals Inflammatory bowel disease Partial lipodystrophy Jejunal diverticulosis with bacterial overgrowth Severe anemia Total parenteral nutrition

PATHOGENESIS OF NAFLD

Day and James proposed “Two hit hypothesis” in 1998





CLINICAL FEATURES OF NAFLD

SYMPTOMS:

Usually they are asymptomatic.NAFLD are detected incidentally, while screening for other disease. Occasionally patient may have right hypochondriac pain, discomfort,associated with fatigue and malaise.

SIGNS:

Hepatomegaly is present in nearly 75% of NAFLD. Other stigmata of liver cell failure like splenomegaly,spider angioma, ascites, may be in late stage of NAFLD when they progress cirrhosis.

LABORATORY FEATURES OF NAFLD

Elevation of liver enzymes are present in nearly 50% of patients with NAFLD. The liver enzymes will be elevated in nearly 80% of patients with advanced NAFLD induced cirrhosis.ALT will be elevated than AST . There will be a rise of 1.5 to 4 fold in liver enzyme level.it will never exceed 10 fold. ANA may be positive in low titer in one fourth of the patients. Elevation of serum ferritin is a marker of advanced cirrhosis [(36)]

LABORATORY FEATURES
Two- to fourfold elevation of serum ALT and AST levels
AST/ALT ratio less than 1 in most patients
Serum alkaline phosphatase level is slightly elevated in one third of patients
Normal serum bilirubin and serum albumin levels and prothrombin time
Elevated serum ferritin level

IMAGING

Ultrasonography of liver may reveal increased echogenicityof liver, it appears as bright liver. USG has a sensitivity of 100% in detecting a fatty liver.

CT: In abdominal computer tomography–fatty liver appears less dense than spleen.

MRI- fatty liver appears bright in T 1 weighted image

For assessing the severity of fatty liver or grading, liver biopsy is needed and is confirmatory for the diagnosis and staging with certainty.

HISTOLOGY OF NNFL AND NASH:

NAFLD is characterized by mixed macro ,micro ,medio vesicular steatosis ,but predominantly macrosteatosis,diffusely distributed ,but perivenular predominance is also noted.

NASH is characterized by hepatitic steatosis as described above, which may correlate with BMI,plus predominantly lobular inflammation with lymphocytes,macrophages,neutrophils is the hallmark of NASH .

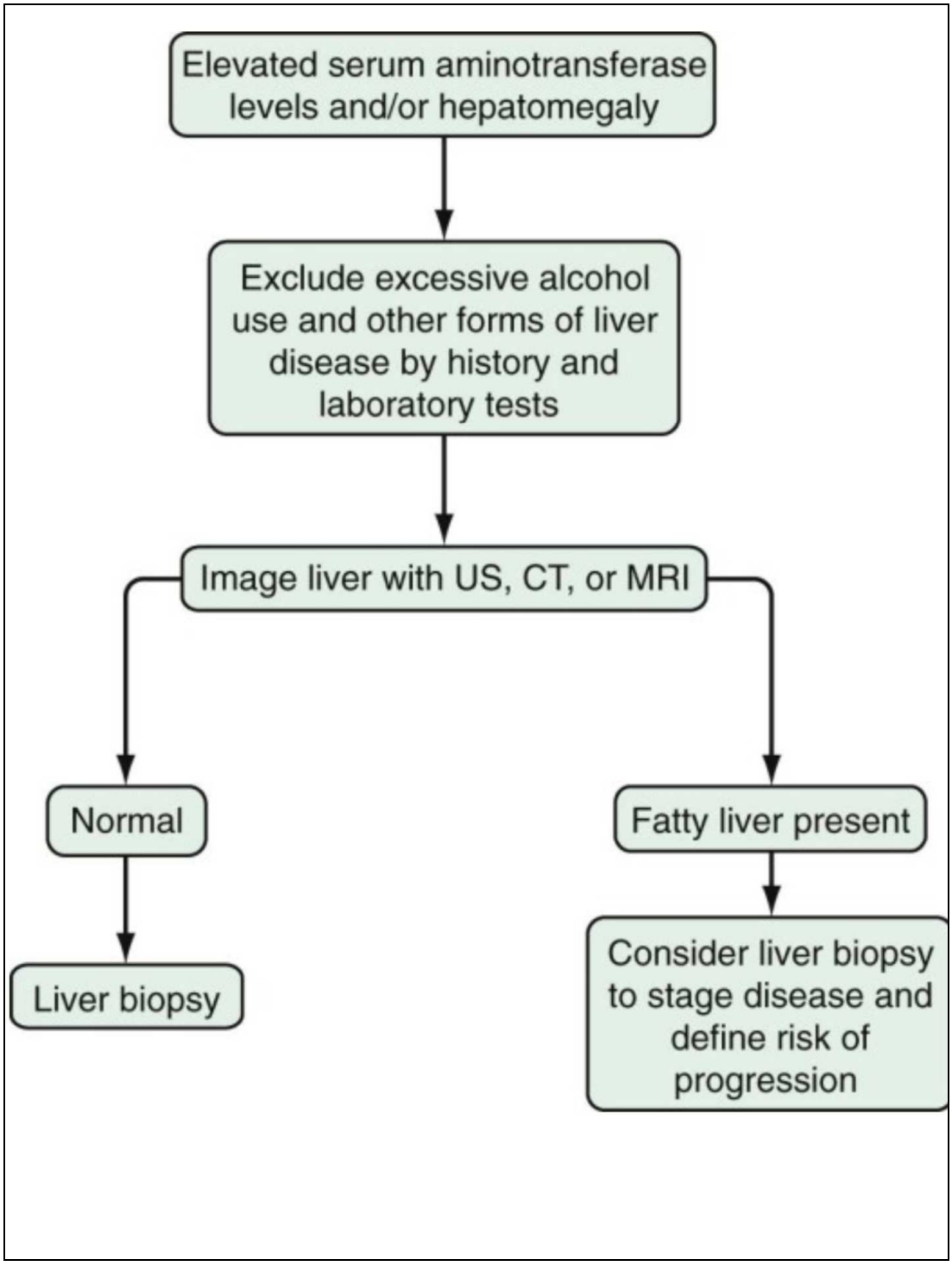
Ballooning of hepatocytes ,presence of "Mallory –Denk bodies" inside the inflamed hepatocytes, Perisinusoidal (zone 3), periportal, perivenular fibrosis may be noticed in advanced NASH .

Present in All or Most Cases
Macrovesicular steatosis
Diffuse or centrilobular steatosis; degree may correlate with BMI
Parenchymal inflammation
Polymorphonuclear neutrophils, lymphocytes, other mononuclear cells
Hepatocyte necrosis
Ballooning hepatocyte degeneration

Observed with Varied Frequencies
Perivenular, perisinusoidal, or periportal fibrosis (37%-84%), moderate to severe in 15%-50%; most prevalent in zone 3 (perivenular)
Cirrhosis (7%-16% on index biopsy specimen)
Mallory bodies
Glycogenated nuclei
Lipogranulomas
Stainable hepatic iron

DIAGNOSIS :

Diagnosis of NAFLD requires both clinical, histologic parameters.A combination of history,physical examination,biochemical parameters ,imaging modality is needed to rule out other causes of steatohepatitis .


Elevated serum aminotransferase levels and/or hepatomegaly
Exclude excessive alcohol use and other forms of liver disease by history and laboratory tests
Image liver with US, CT, or MRI
Normal
Liver biopsy
Fatty liver present
Consider liver biopsy to stage disease and define risk of progression

CLASSIFICATION

LIVER BIOPSY: is a invasive, procedure with minimal complication.

WORKING CLASSIFICATION OF

NAFLD

NNFL
Type 1 NAFLD: Steatosis with no inflammation or fibrosis
Type 2 NAFLD: Steatosis with non-specific lobular inflammation but absent of fibrosis or hepatocyte ballooning
NASH
Type 3 NAFLD: Steatosis with inflammation and fibrosis of variable levels (NASH)
Type 4 NAFLD: Steatosis, inflammation, hepatocyte ballooning, and fibrosis or Mallory–Denk bodies (NASH)

FIBROSIS STAGING OF NONALCOHOLIC STEATOHEPATITIS (NASH):

Stage 1: **Pericentral vein or sinusoidal fibrosis (Zone 3)**
Stage 2: **Sinusoidal (Zone 3) and periportal fibrosis (Zone 1)**
Stage 3: **Bridging fibrosis between Zone 3 and Zone 1**
Stage 4: **Cirrhosis**
NASH with cirrhosis
Cirrhosis with features suggestive of NASH
Non-specific (cryptogenic) cirrhosis

NON INVASIVE MARKERS OF FIBROSIS IN NAFLD

Hematological parameters to estimate hepatic fibrosis includes total bilirubin, GGTP levels, apolipoprotein A-1, serum α2-macroglobulin, haptoglobin,serumdehydroepiandrosteronelevelsand serum hyaluronic acid levels.

FIBROTEST:

Detects bridging fibrosis and cirrhosis a value >0.7 has a specificity of 98% for advanced fibrosis. [35]

Fibroscan (transient elastography):

Quantifies liver stiffness and fibrosis.

NAFLD Fibrosis Score developed byAngulo et al,incorporates age, BMI, hyperglycemia, AST/ALT ratio, platelet count, and serum albumin level is yet another noninvasive method to quantify fibrosis.

COURSE OF NAFLD:

The course of NAFLD is favorable if there is no hepatic fibrosis, or cirrhosis. Few patients of NAFLD can progress to liver failure, cirrhosis (3%) , hepatocellular carcinoma.

RISK FACTORS FOR ADVANCED NAFLD

Clinical
Older age (>50 years)
Obesity
Diabetes mellitus/insulin resistance
Hypertension
Laboratory
AST/ALT ratio > 1
Serum ALT level > twice the upper limit of normal
Serum triglyceride levels > 155 mg/dL
Histologic
Severe steatosis
Necroinflammatory activity (hepatocyte ballooning, necrosis)
Stainable iron

TREATMENT OF NAFLD

Avoidance of toxins
- Discontinue potentially offending medications/toxins
- Minimize alcohol intake

Exercise and diet
- Moderate, sustained exercise and weight loss in overweight patients
- Effects of specific diets are not known

Antidiabetic/insulin-sensitizing agents
- Metformin
- Thiazolidinediones

Lipid-lowering agents
- Gemfibrozil
- Statins

Antioxidants
- Betaine
- *N*-acetylcysteine
- Superoxide dismutase
- Vitamin E

Iron reduction by phlebotomy

Inflammatory mediators by:
- Agents that affect increasing mitochondrial ATP stores and/or activity
- Agents that affect modulating leptin activity
- Agents that affect modulating TNF-α activity
- Agents that affect raising adiponectin levels

Bariatric surgery for morbid obesity

METABOLIC SYNDROME:

Metabolic syndrome comprises of cluster of metabolic risk factors which predisposes a person for cardiovascular disease. It was Reaven in 1988 who observed this cluster of risk factors and he labeled these multiplex risk factors as syndrome X. Other terminologies used for this syndrome are insulin resistance syndrome, metabolic syndrome, and deadly quartet.

COMPONENTS OF METABOLIC SYNDROME

i) Raised blood pressure

ii) Atherogenic dyslipidemia

iii) Abdominal obesity

iv) Insulin resistance glucose intolerance

v) Prothrombotic state

vi) Proinflammatory state

Various criteria's are put forth and consistently revised by 4 organizations to identify individuals with this syndrome, for primary prevention of cardiovascular diseases, namely

I. International Diabetes Federation (IDF)

II. National Cholesterol Education Program's Adult Treatment Panel III (ATP III)

III.WHO Clinical Criteria for Metabolic Syndrome

IV. American Association of Clinical Endocrinologists (AACE) Clinical Criteria for diagnosis of the Insulin Resistance Syndrome

V. European group for the study of insulin resistance (EGIR)

CRITERIA FOR METABOLIC SYNDROME

INTERNATIONAL DIABETES FEDERATION (IDF):

Central obesity

Based on waist circumference and adjusted according to the ethinicity of patient plus any two of the following

Raised triglycerides	≥ 150 mg/dL (1.7 mmol/L) **or specific treatment for this lipid abnormality**
Reduced HDL cholesterol	< 40 mg/dL (1.03 mmol/L) in males < 50 mg/dL (1.29 mmol/L) in females **or specific treatment for this lipid abnormality**
Raised blood pressure	systolic BP ≥ 130 or diastolic BP ≥ 85 mm Hg **or treatment of previously diagnosed hypertension**
Raised fasting plasma glucose	(FPG) ≥ 100 mg/dL (5.6 mmol/L), **or previously diagnosed type 2 diabetes** If above 5.6 mmol/L or 100 mg/dL, OGTT is strongly recommended but is not necessary to define presence of the syndrome.

WAIST CIRCUMFERENCE BASED ON ETHINICITY

Ethnic group	Male(cm)	Female(cm)
South Asia	≥90	≥80
Chinese	≥90	≥80
Europe	≥94	≥80
Japanese	≥90	≥80
South and central America, African, eastern mediaterranean	As per south asia	

NATIONAL CHOLESTEROL EDUCATION PROGRAM'S ADULT TREATMENT PANEL III (ATP III)

Any three or more of the following:

Risk factors	**Defining level**
Central obesity Male Female	Waist circumference(cm) >102 >88
Blood pressure	≥ 130/≥85mmHg
Triglycerides	≥ 150mg/dL(1.7 mmol/L)
HDL cholesterol Men female	 <40mg/dL(1.03mmol/L) <50mg/dL (1.29mmol/L)
Fasting glucose	≥110mg/dL(6.1 mmol/L)

WHO CRITERIA FOR METABOLIC SYNDROME

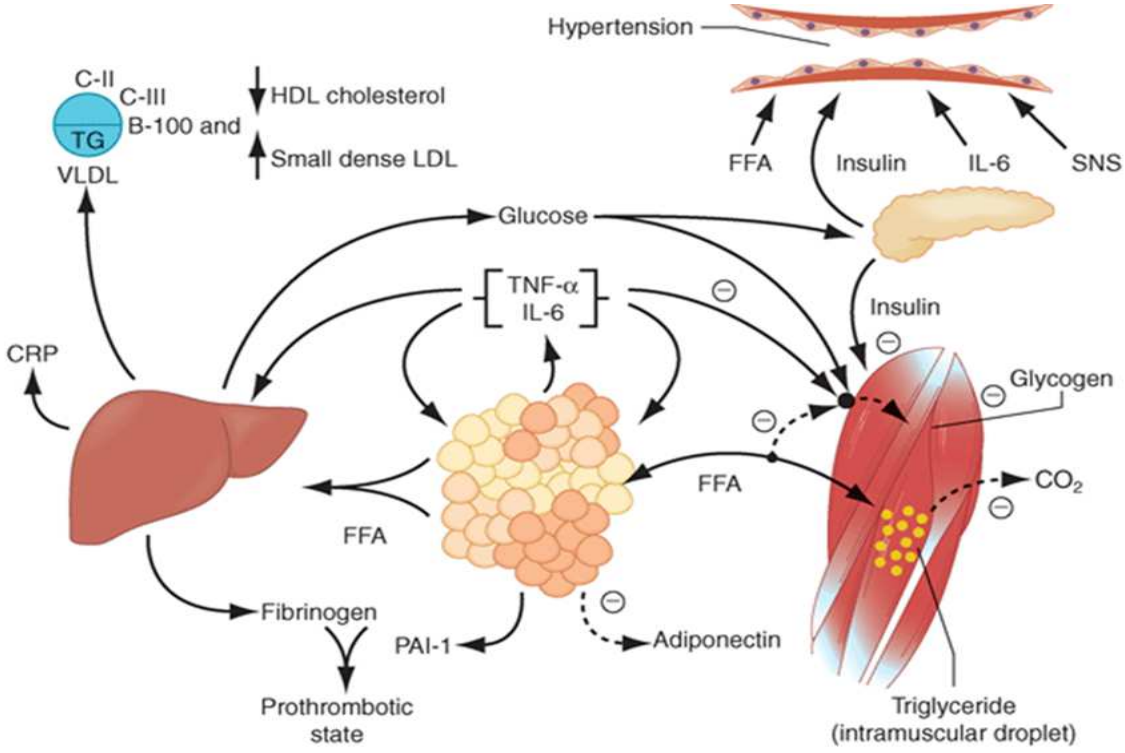
Impaired glucose regulation or diabetes mellitus

Presence of insulin resistance (hyperinsulinemic glycemic condition)

Plus two ormore of the following:

Risk factors	**Defining level**
Central obesity Male Female	waist –hip ratio >0.90 >0.85 or BMI>30 kg/m^2
Blood pressure	≥140/90mmHg
Triglycerides	≥ 150mg/dL(1.7 mmol/L)
HDL cholesterol Men female	 35mg/dL (0.9mmol/L): 39mg/dL (<1.0mmol/L)
1) Microalbuminuria	urine albumin excretion rate ≥20g/min albumin : creatinine ratio >30mg/g

PATHOGENESIS OF METABOLIC SYNDROME:



Obesity and insulin resistance are responsible for the components of metabolic syndrome and hence risk for CVD, and type 2 diabetes.

EFFECT OF OBESITY AND INSULIN RESISTANCE:

Obesity leads to excess of adipose tissue and when insulin resistance occurs there is impaired antilipolytic activity of insulin. This result in increased lipolysis and increase in free fatty acid, which gets deposited in liver and muscle .other effects of insulin resistance include decreased insulin mediated glucose uptake in liver ,muscle, increased triglyceride and

lipoprotein formation like VLDL,LDL in liver .increased glucose and FFA in circulation results hyperinsulinemic state .

Increased reabsorption of sodium and increased sympathetic nervous system activity due to hyperinsulinemiaresults in hypertension in metabolic syndrome.

EFFECTS OF CYTOKINES

IL-6 ,TNF alpha are produced from the excess adipose tissue .this enhances the insulin resistance ,increases conversion of triglyceride to FFA, hepatic production of glucose, CRP, fibrinogen, adipocyte production of PAI resulting in a proinflammatory and prothrombotic state respectively .

Reduced levels of anti-inflammatory, insulin sensitizing cytokine adiponectin also predisposes to metabolic syndrome.

Role of adiponectin in metabolic syndrome: Adiponectin is an antiinflammatory cytokine produced exclusively by adipose tissue. It increases the insulin sensitivity, reduces the rate of glucose production by liver.in muscle it enhances the glucose uptake, fatty acid oxidation. Reduced adiponectin may contribute to the development of metabolic syndrome and it correlates with the development of metabolic syndrome.

RISK FACTORS FOR METABOLIC SYNDROME

1) Overweight/obesity
2) Sedentarylifestyle
3) Diabetes mellitus
4) Coronary artery disease
5) Lipodystrophy
6) Aging

CLINICAL FEATURES OF METABOLIC SYNDROME

On examination signs which should alert a clinician for features of metabolic syndrome includes elevated blood pressure, increased waist circumference lipodystrophy, acanthosis nigricans and features of other associated diseases due to metabolic syndrome.

ASSOCIATED DISEASE

Cardiovascular disease, type 2 diabetes, nonalcoholic fatty liver disease , hyperuricemia, polycystic ovary syndrome, obstructive sleep apnea syndrome.

DIAGNOSIS

An individual must satisfy the criteria for metabolic syndrome based on examination and laboratory parameters.

Biomarkers for supporting the diagnosis and research in metabolic syndrome:

BIOMARKERS FOR SUPPORTING DIAGNOSIS AND RESEARCH IN METABOLIC SYNDROME

Measuring abnormal body fat distribution	Adipose tissue marker- leptin, adiponectin Liver fat distribution-MRS General body fat distribution-DEXA Abdominal fat distribution-CT/MRI

Atherogenic dyslipidaemia	Apo B, (non HDL c),,small LDL particles
Glycemic status	OGTT
Insulin resistance	Fasting insulin /proinsulin levels, HOMA-IR Free fatty acid level (fasting and post prandial) Insulin resistance by Bergman minimal model M value from clamp Uric acid
Prothrombotic state	Fibrinolytic factors-PAI Clotting factors-fibrinogen
Proinflammatory state	High sensitivity C-reactive protein IL-6,TNF alpha, Adiponectin
Vascular dyregulation	Microalbuminuria

Other tests which supports the diagnosis includes liver function test, uric acid level, sleep study if patient has obstructive sleep apnea.

PREVENTION OF CVD

LIFESTYLE MODIFICATION AND WEIGHT REDUCTION

Physical activity of 60–90 min of daily activity is recommended.indifficult situations at least 30 min of moderate-intensity daily activity is recommended .this has proved to reduce the blood pressure, serum cholesterol, triglycerides levels, PAI, CRP levels, insulin resistance , increases HDL level.

DIET:

High-quality diet with high quantity of fruits, vegetables, whole grains, lean poultry, and fish. Diets restrictedin carbohydrates, saturated fats (<7% of calories), trans-fats (as few as possible), and cholesterol (<200 mg daily)are advised.

OBESITY

Appetite suppressants -phentermine, sibutramine can be used on short term , fat absorption inhibitor-orlistat are found to reduce the incidence of diabetes. Bariatric surgery ,gastric bypass surgery can be considered in individuals with BMI >35-40kg/m^2.

DYSLIPIDEMIA

LDL CHOLESTEROL

Targeted LDL cholesterol goal is <130 mg/dL in patients with history of CVD the goal is <100 mg/dL .in uncontrolled individuals drugs like statins, cholesterol absorption inhibitor ezetimibe can be used. Fibrates especially fenofibrate is used when both LDL cholesterol and triglcerides are elevated. Nicotinic acid has modest benefit in lowering LDL cholesterol. Bile acid sequestrants cholestyramine and colestipol can be when LDL cholesterol alone is elevated as it may increase the triglycerides.

TRIGLYCERIDES:

Fibrates are the drug of choice for lowering the triglyceride level. other drugs which can lower triglycerides include statins, nicotinic acid, and high doses of omega-3 fatty acids.

HDL CHOLESTEROL:

Nnicotinic acid is the only drug which can raise the HDL cholesterol level.

Drugs that reduces insulin sensitivity includes metformin and thiazolidinediones.

HYPERTENSION

Antihypertensive drugs as per hypertension guidelines should be prescribed. ACE inhibitors or ARB‘s are preferred for hypertensive without diabetes. Low dose aspirin as antithrombotic are other drugs which are proved to reduce the CVD and can be used as primary and secondary prevention.

NEWER GROUP OF DRUGS IN CLINCSL TRIAL:

PPAR agonists interacting with both alpha and gamma receptors, incretin mimics, protein tyrosine phosphatase IB inhibitors, endocannabinoid receptor inhibitor, dipeptidyl peptidase IV inhibitors are under trial.

Monitoring of individuals with metabolic syndrome:

MATERIALS AND METHODS

MATERIALS AND METHODS

SELECTION OF PATIENTS:

All subjects were explained about the psoriasis and its comorbidities.

All patients were informed about the proceedings of the study and the advantage of participating in the study in their own language.

Consents were obtained from all the subjects before participating in the study

STUDY DESIGN:

Cross sectional study

STUDY CENTRE:

Institute of Dermatology Venereology and Leprosy,

Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai

DURATION OF THE STUDY

6 months April 2014 toSeptember 2014

INCLUSION CRITERIA

Patients diagnosed with psoriasis, aged>20 years, who are willing to participate in the study were included.

EXCLUSION CRITERIA

i) Psoriasis patients on hepatotoxic drugs, such as methotrexate, corticosteroids, psoralens, acitretin, oral contraceptive pills, tamoxifen, antituberculous drugs.

ii) Patients with significant alcohol use Consumption of more than 20 to 40 g of alcohol.(2 standard drinks in female and 3 standard drinks in male per day).

iii) Patients who are not willing to participate in our study.

iv) H/S/O hemochromatosis, Wilson's disease,

SAMPLE SIZE

A total of 165 patients were included in the study.

.

DATA COLLECTION METHOD

All patients included in our study are subjected to a thorough

1. Clinical history,

2. General examination,

3. Clinical examination,

4. Dermatologicalexamination,

5. Blood investigation,

6.Ultrasonography by Radiologist

METHODOLOGY:

HISTORY:

A detailed history on psoriasis and H/S/O any comorbid illness were collected.

The particulars include,

Age

Sex

Duration of psoriasis

Site of involvement

H/O arthritis

H/O Diabetes mellitus, hypertension, abdomen symptoms like pain, discomfort, jaundice.

Treatment history

H/O Alcohol consumption if present- type, quantity, and duration of alcohol intake.

GENERAL EXAMINATION

For all subjects the following parameters were assessed.

1. BMI based on weight and height using the formula weight/height in meters ^{2}

2. Waist circumference was measured by placing a measuring tape around the abdomen at the level of the iliac crest, firmly.

3. Blood pressure was measured in sitting posture on the right arm.

4. Other general examination was done in all subjects.

SYSTEMIC EXAMINATION

The following systems were examined

Abdomen: patients were examine for the presence of organomegaly especially hepatomegaly.

Cardiovascular system

Central nervous system

Respiratory system.

DERMATOLOGICAL EXAMINATION

All confirmed cases of psoriasis either clinically or based on histopathology were examined and data collected on

1. Type of psoriasis

2. Site of involvement

3. Presence of psoriatic arthritis

4. PASI calculated to assess the severity

PROCEDURE / INVESTIGATION DETAILS:

Patients will be subjected to blood investigations like

- Liver function tests including
  - AST, ALT,
- Fasting blood glucose
- Fasting lipid profile which includes
  - Serum triglycerides
  - Serum cholesterol
  - Serum High density lipoprotein.

Based on the above parameters, the presence of metabolic syndrome is identified using , INTERNATIONAL DIABETES FEDERATION(IDF)

All patients are subjected to ultra sonogram of the abdomen in empty stomach

Patients who are found to have nonalcoholic fatty liver by USG, are subjected to anti HCV,

HBs Ag

ANALYSIS PLAN

Statistical analysis was done using SPSS version 17.0

SPONSORSHIP :

NO

CONFLICT OF INTEREST :

None

OBSERVATION

OBSERVATION AND RESULTS

A total of 165 patients participated in our study. Of them 98 (59.3%) are males and 67(40.6%) are females .They are distributed in the age group from 20 years to 72 years .All the subjects are categorized based on the severity by PASI score and analyzed. Our patients had PASI score ranging from as low as 5 to 60.Majority of the subjects are males in the age group 30 to 50. Majority of them belonged to PASI score of 11-30 group.

FIGURE 1

DISTRIBUTION OF PATIENTS ACCORDING TO PASI

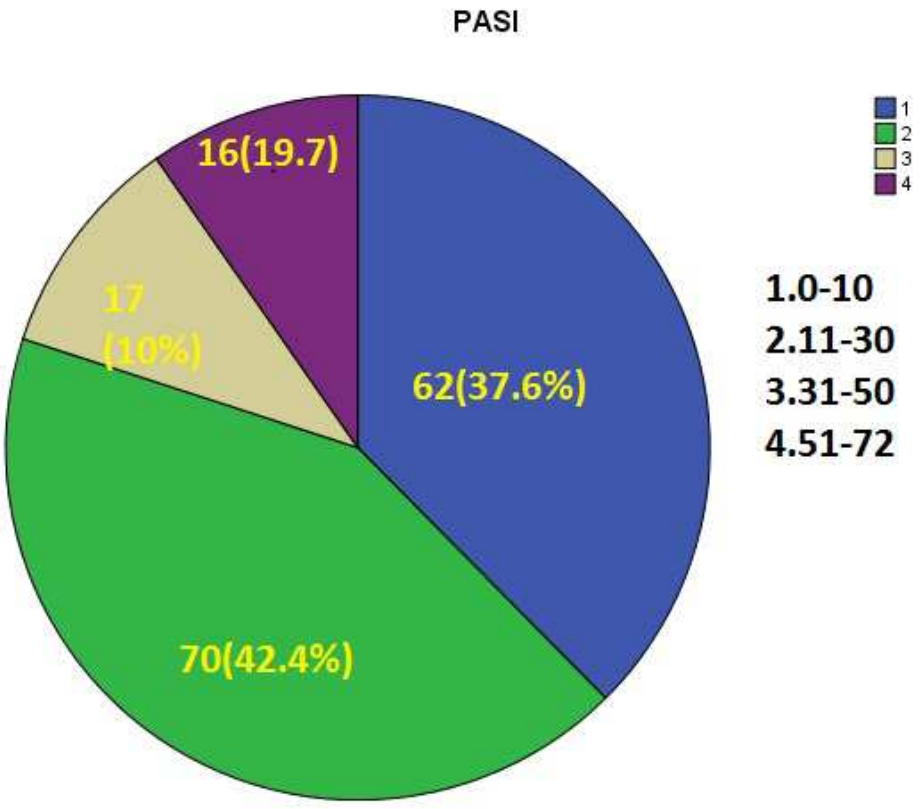
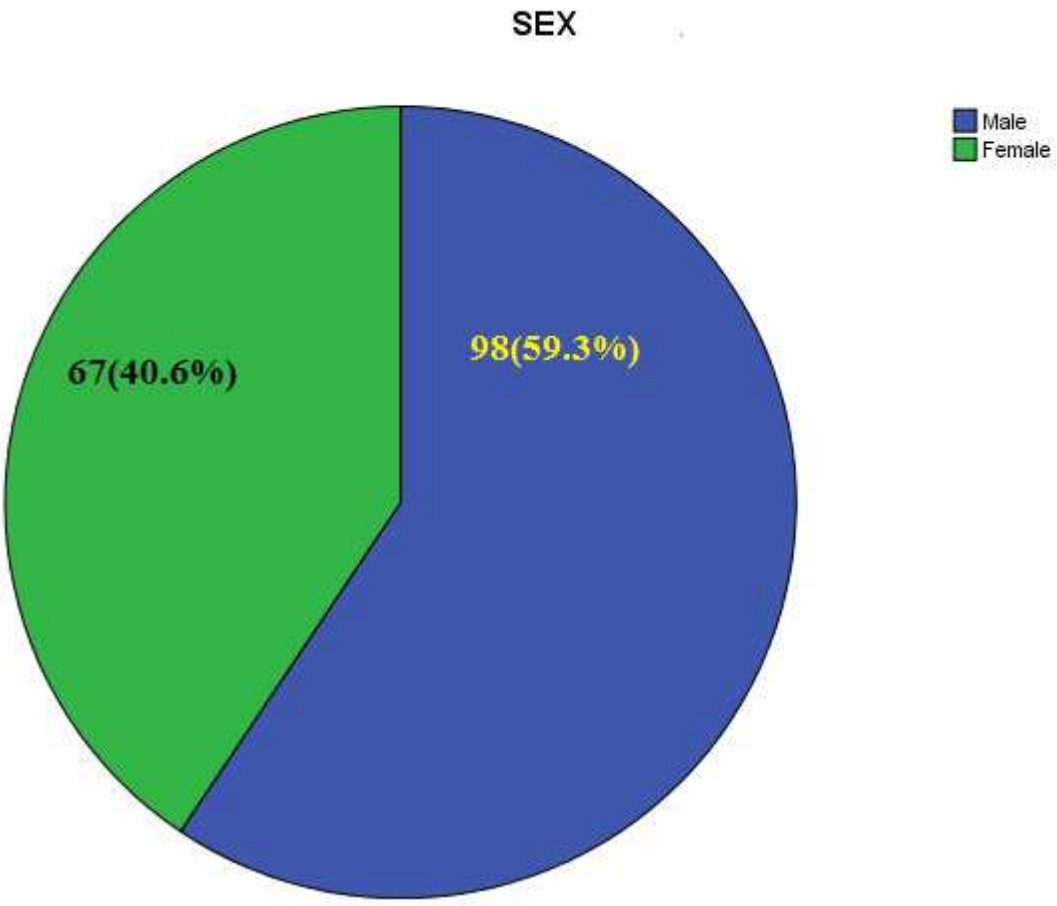


FIGURE 2

SEX WISE DISTRIBUTION OF PSORIASIS PATIENTS



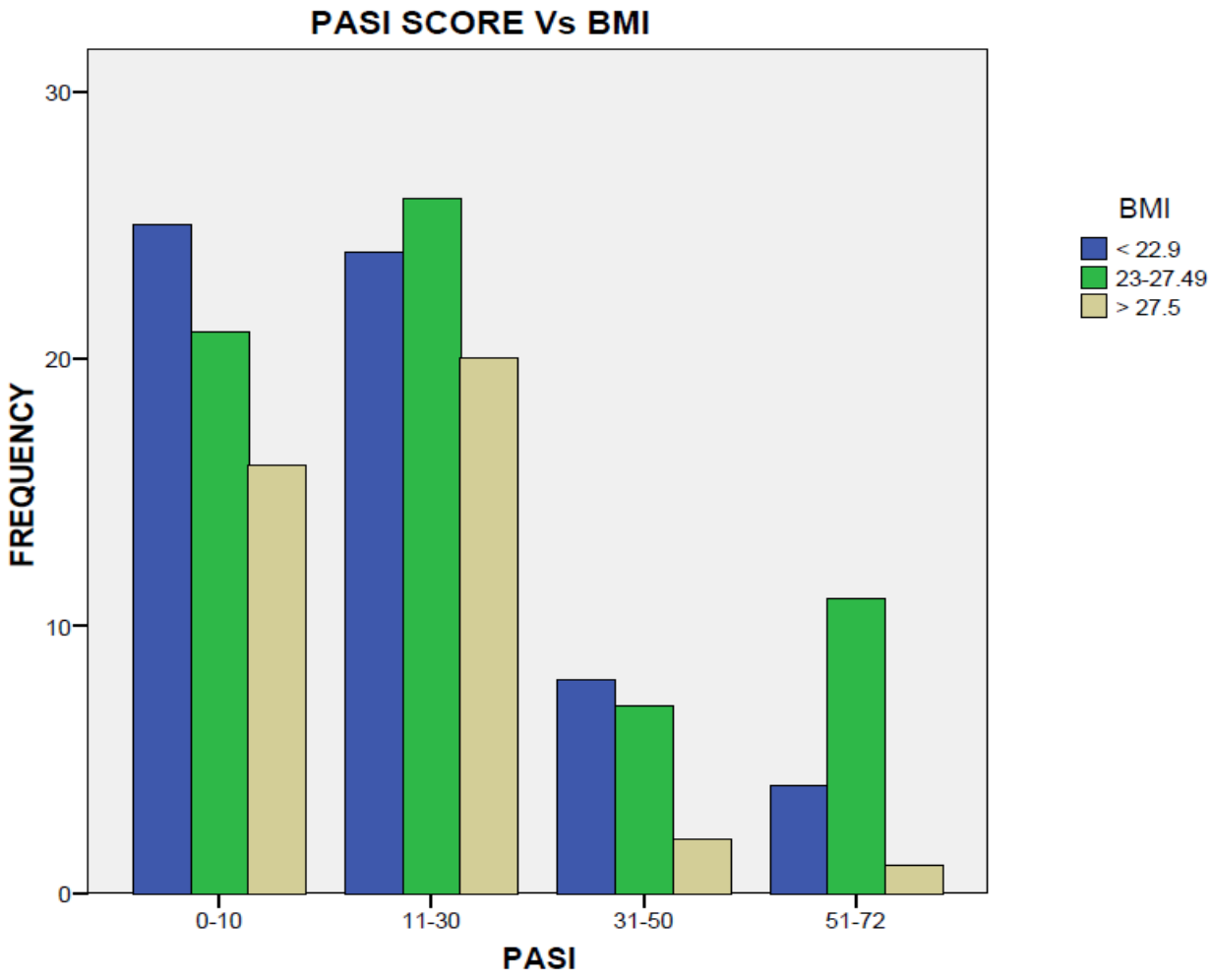
OBESITY:

TABLE 1

PSORIASIS PATIENTS AND BODY MASS INDEX

PASI	NO.OF PATIENTS			TOTAL
	<22.9	23-27.49	>27.5	
0-10	25(41%)	21(32.3%)	16(41%)	62(37.6%)
11-30	24(39.3%)	26(40%)	20(51.3%)	70(42.4%)
31-50	8(13.1%)	7(10.8%)	2(5.1%)	17(10.3%)
51-72	4(6.6%)	11(16.9%)	1(2.6%)	16(9.7%)
TOTAL	61(37%)	65(39%)	39(24%)	16(95)

FIGURE 3

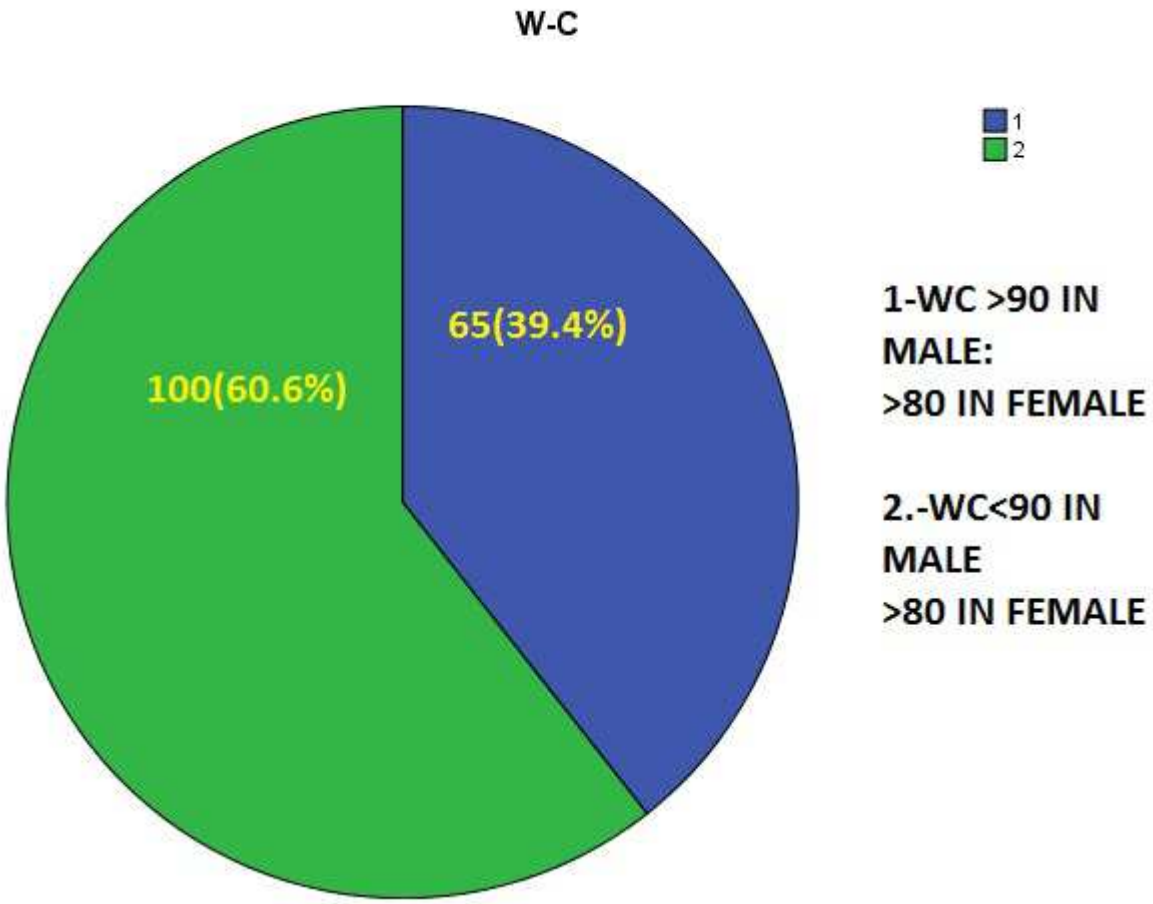


39(24%) of our patients are obese, and patients belonging to PASI score 11-30 years had increased BMI

Waist circumference based on IDF criteria ,39.4% of patients are found to have central obesity.

FIGURE 4

DISTRIBUTION BASED ON WAIST CIRCUMFERENCE

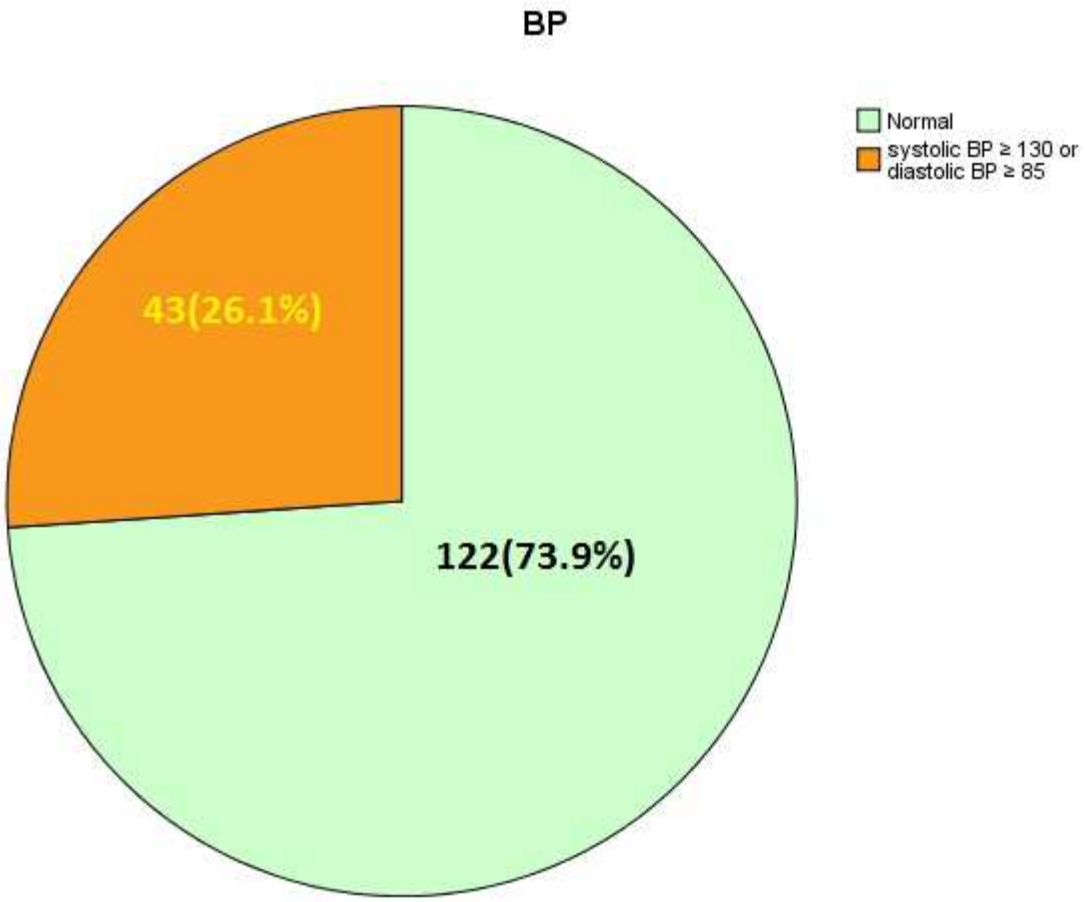


HYPERTENSION

43(26.1%) of 165 patients had hypertension, of which 12 out of 165 patients are newly detected to have hypertension , and the remaining are already on treatment. Of these only 6 are < 40 years , and the rest are >40 years.

FIGURE 5

HYPERTENSION IN OUR STUDY

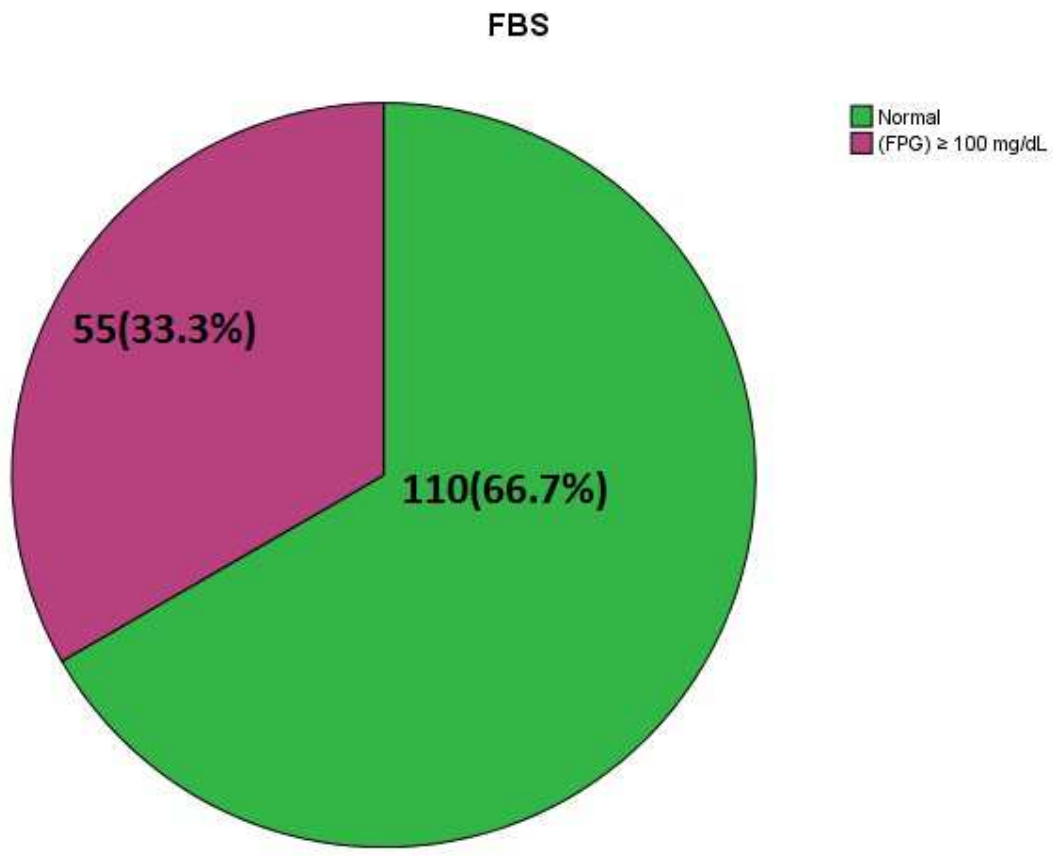


FASTING BLOOD GLUCOSE

55(33.3%) out of 165 had hyperglycemia. Of them 8 are newly detected. Remaining patients are on medications.

FIGURE 6

HYPERGLYCEMIC STATUS



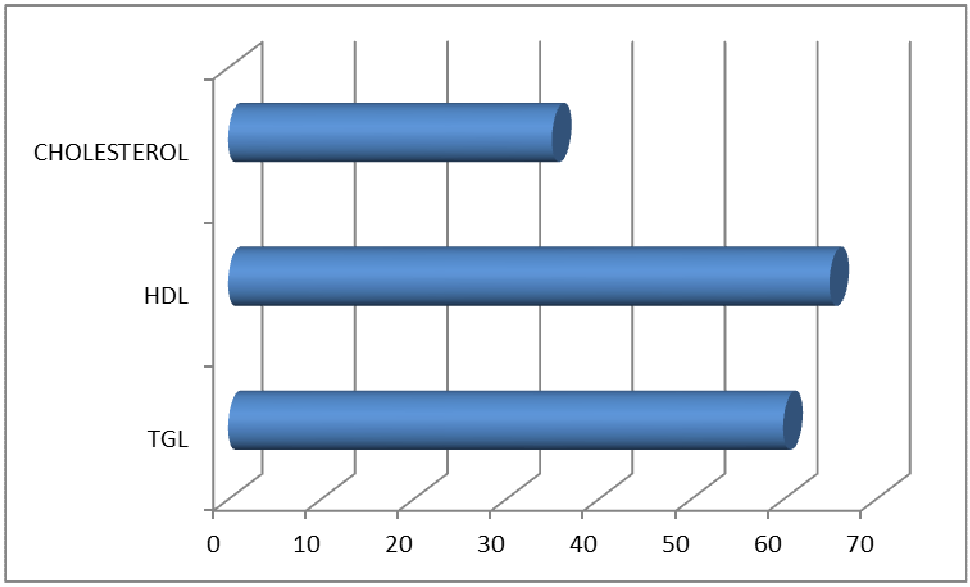
DYSLIPIDEMIA:

Fasting lipid profile is deranged in large number of patients significantly. Triglycerides , cholesterol are elevated in 60(36%) and 35(21%)patients.

Whereas reduced HDL is found in 65(39%) patients.

FIGURE 7

DYSLIPIDEMIA IN PSORIASIS



LIPOPROTEIN LEVELS IN OUR STUDY

FIGURE 8

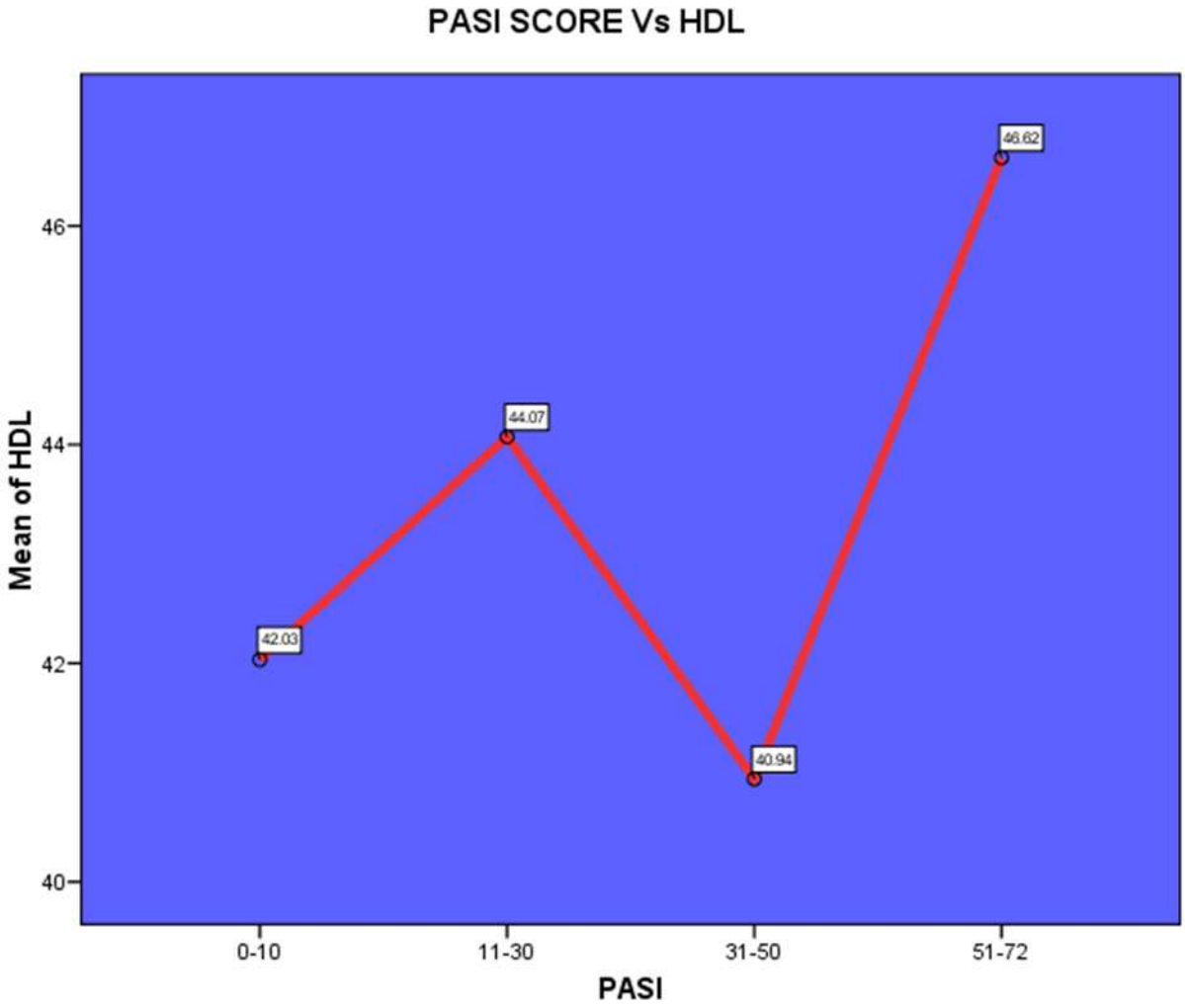


FIGURE 9

TRIGLYCERIDE LEVELS IN OUR PATIENTS

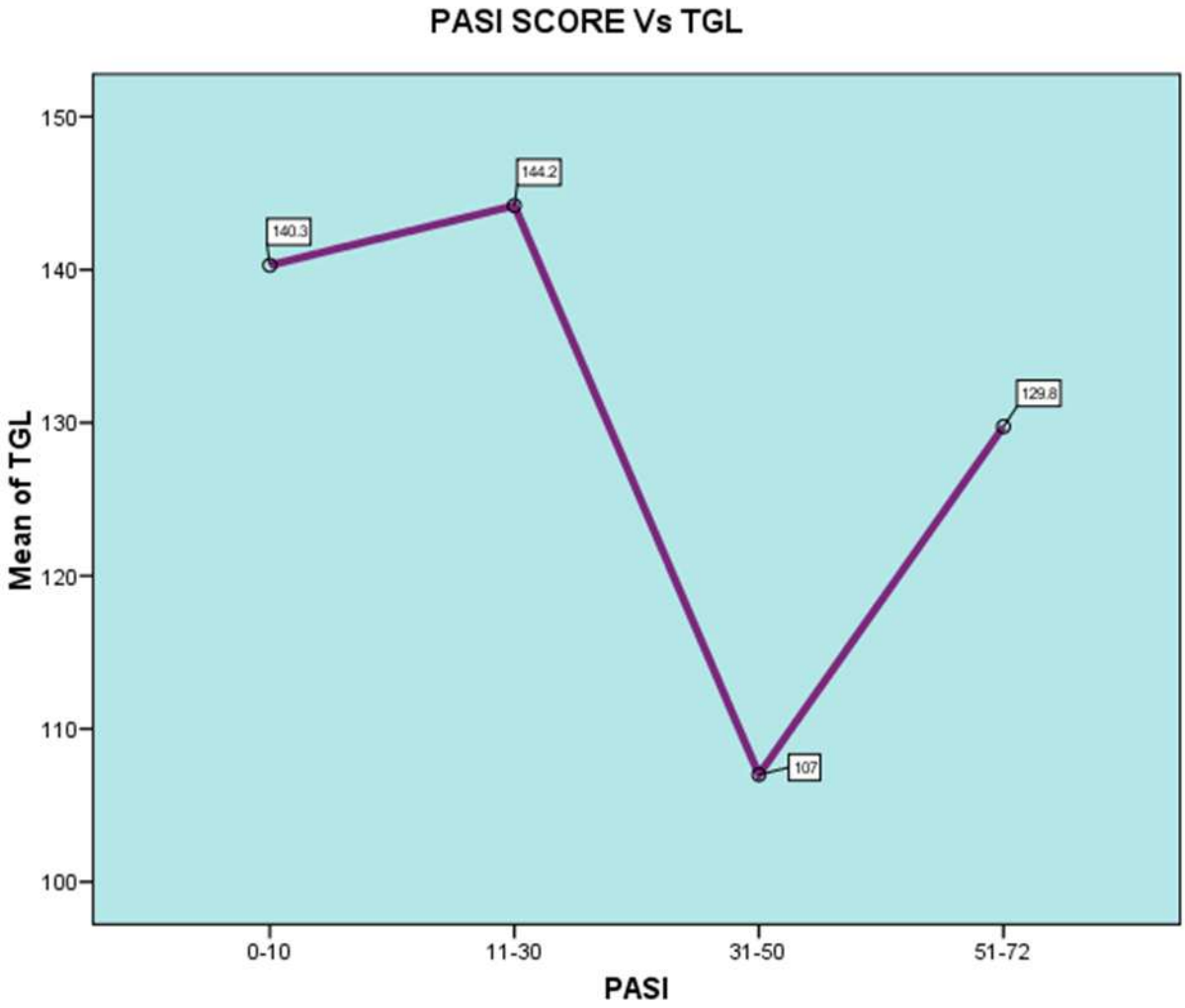
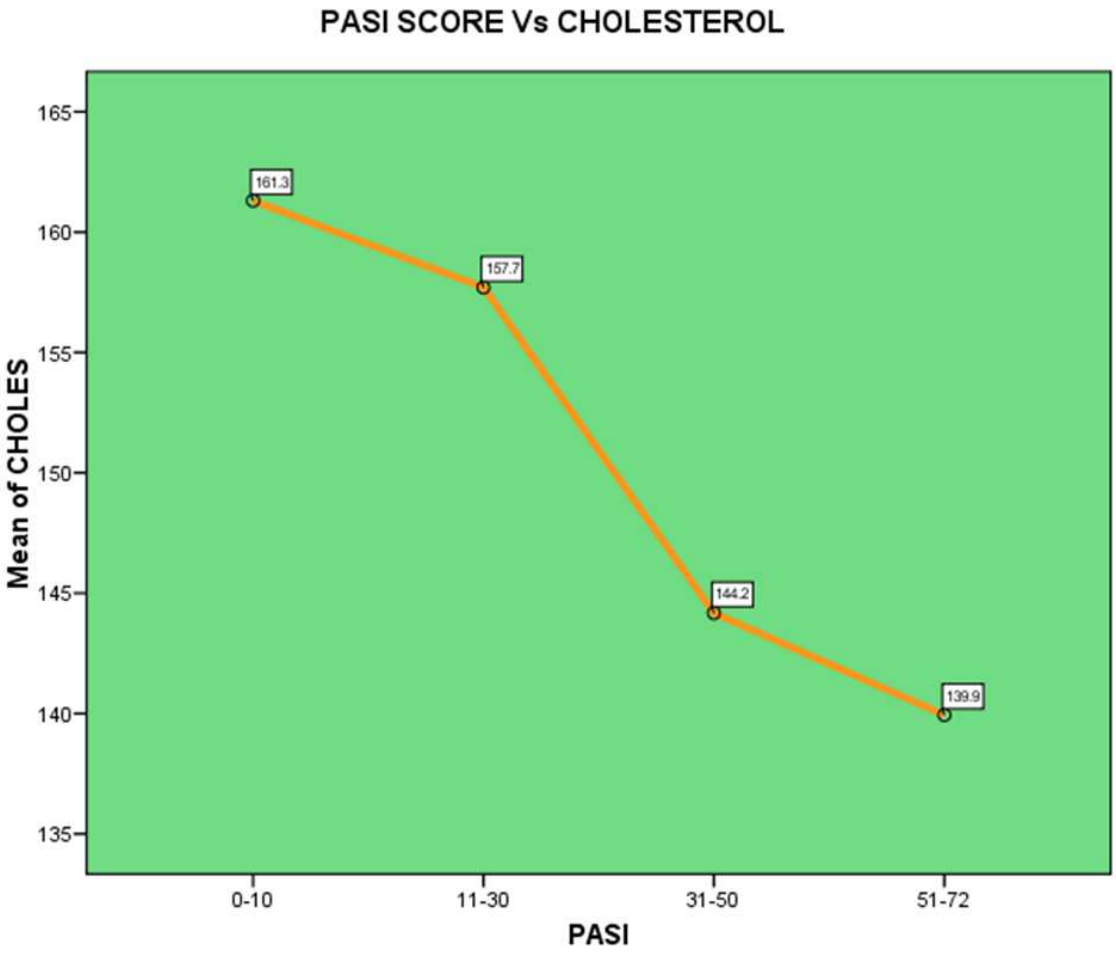


FIGURE 10

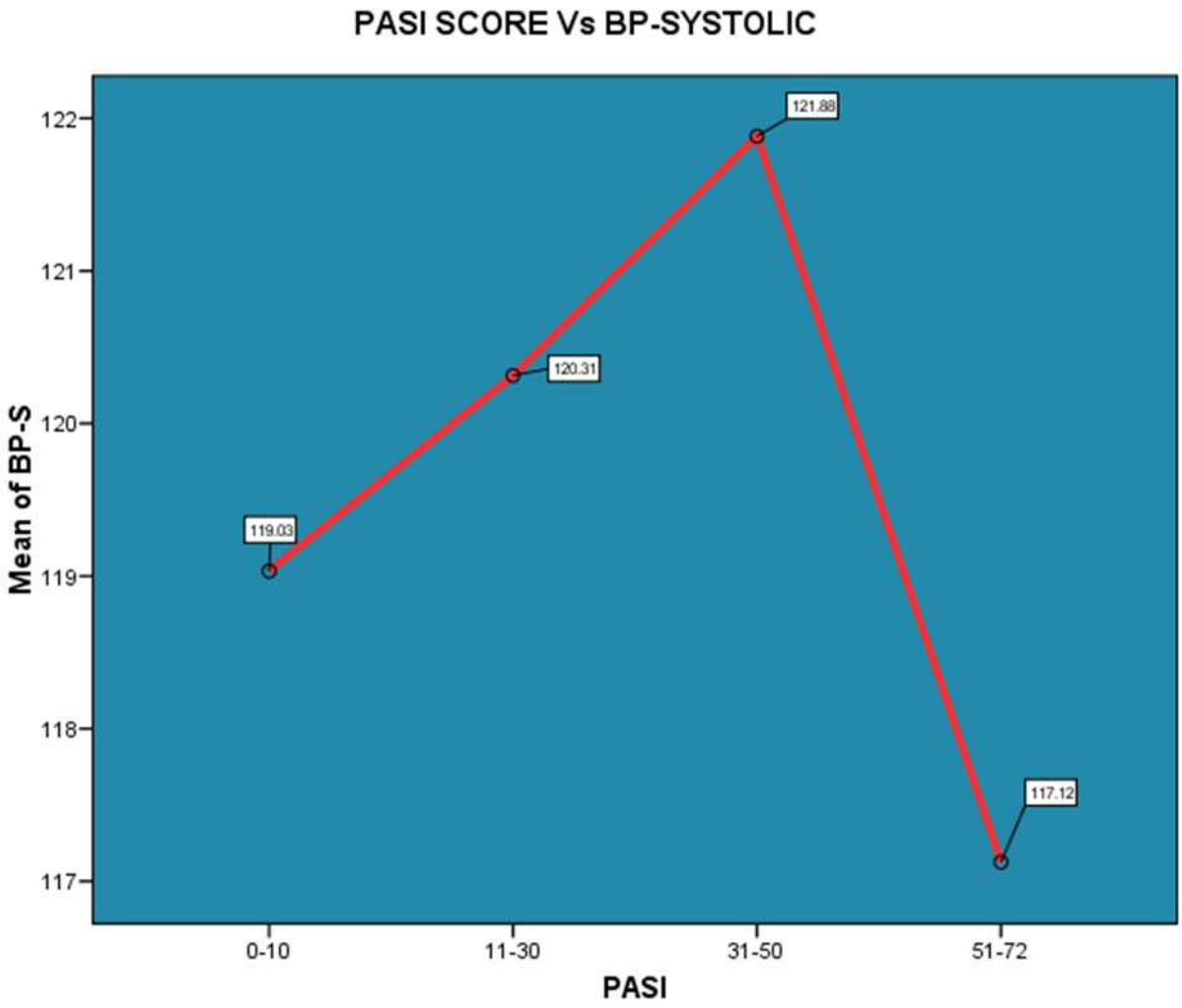
CHOLESTEROL LEVELS IN OUR PATIENTS

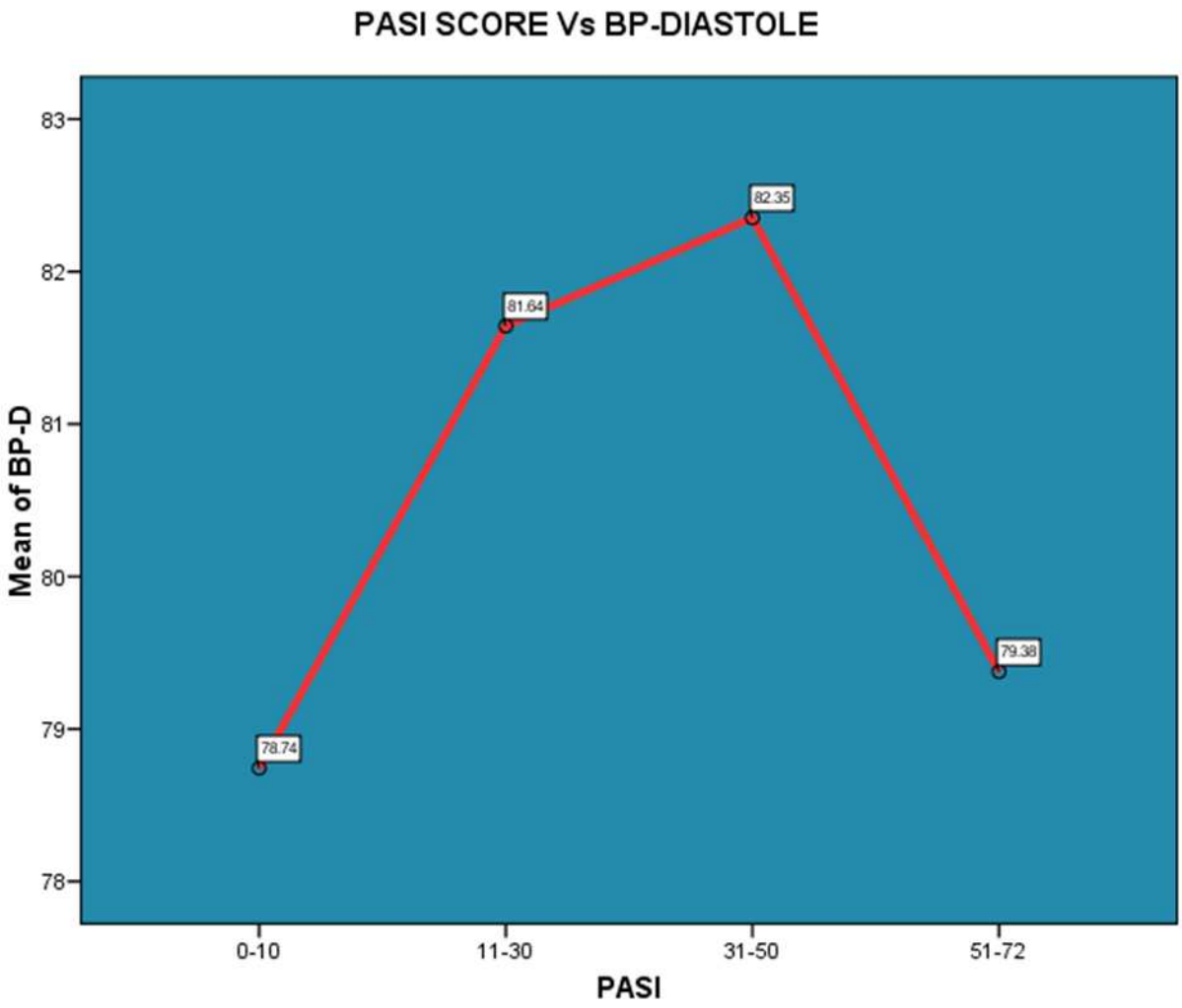


SYSTOLIC AND DIASTOLIC BLOOD PRESSURE

IN OUR STUDY

FIGURE 11




PASI SCORE Vs BP-DIASTOLE
Mean of BP-D
83
82
81
80
79
78
82.35
81.64
79.38
78.74
0-10
11-30
31-50
51-72
PASI

PSORIASIS NONALCOHOLIC FATTY LIVER:

On Ultrasonography around 75(45.5%) are found to have NAFLD. The prevalence of NAFLD is estimated as 45.5%.

Of these patients 16 had metabolic syndrome

They are present predominately in 11-30 PASI score.

They were distributed equally in all age groups, and in duration of illness.

FIGURE 12

PREVALENCE OF NON ALCOHOLIC FATTY LIVER

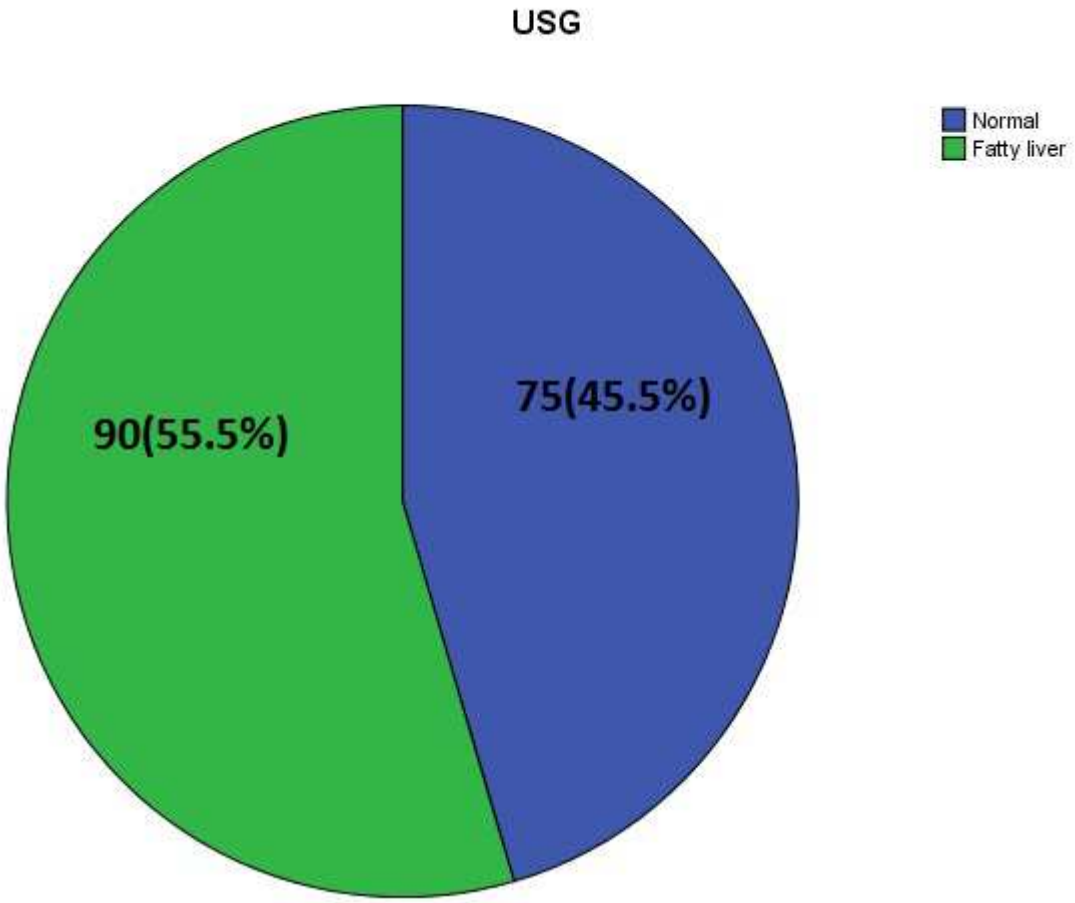
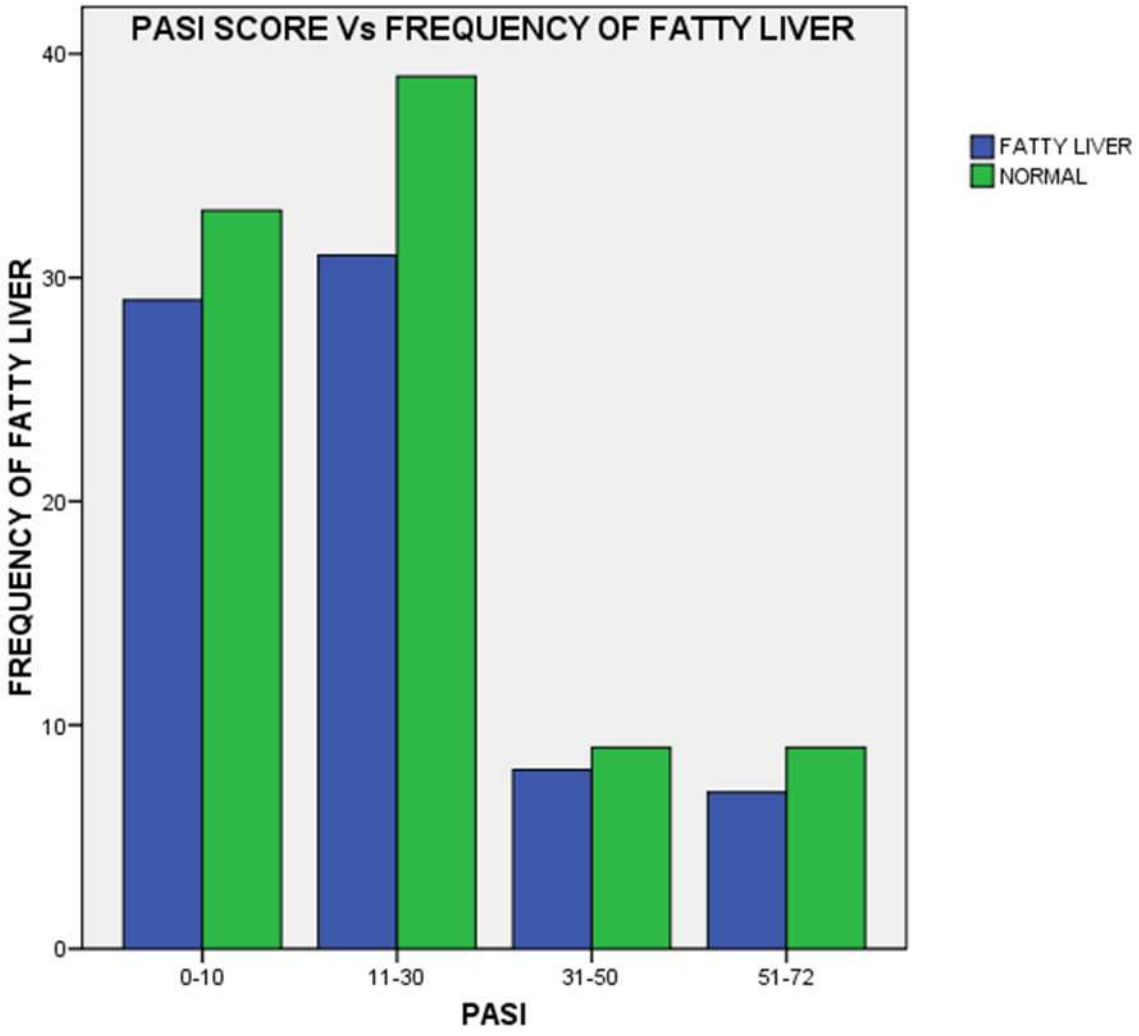
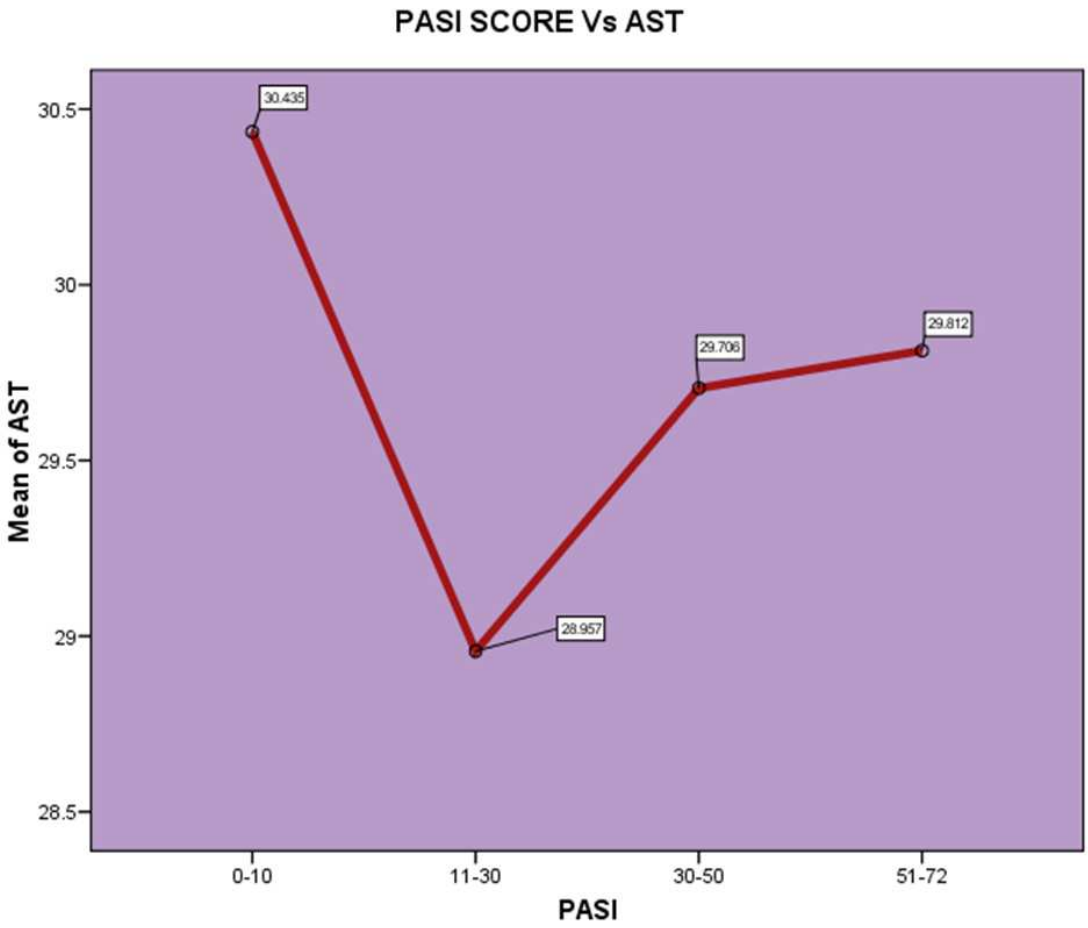


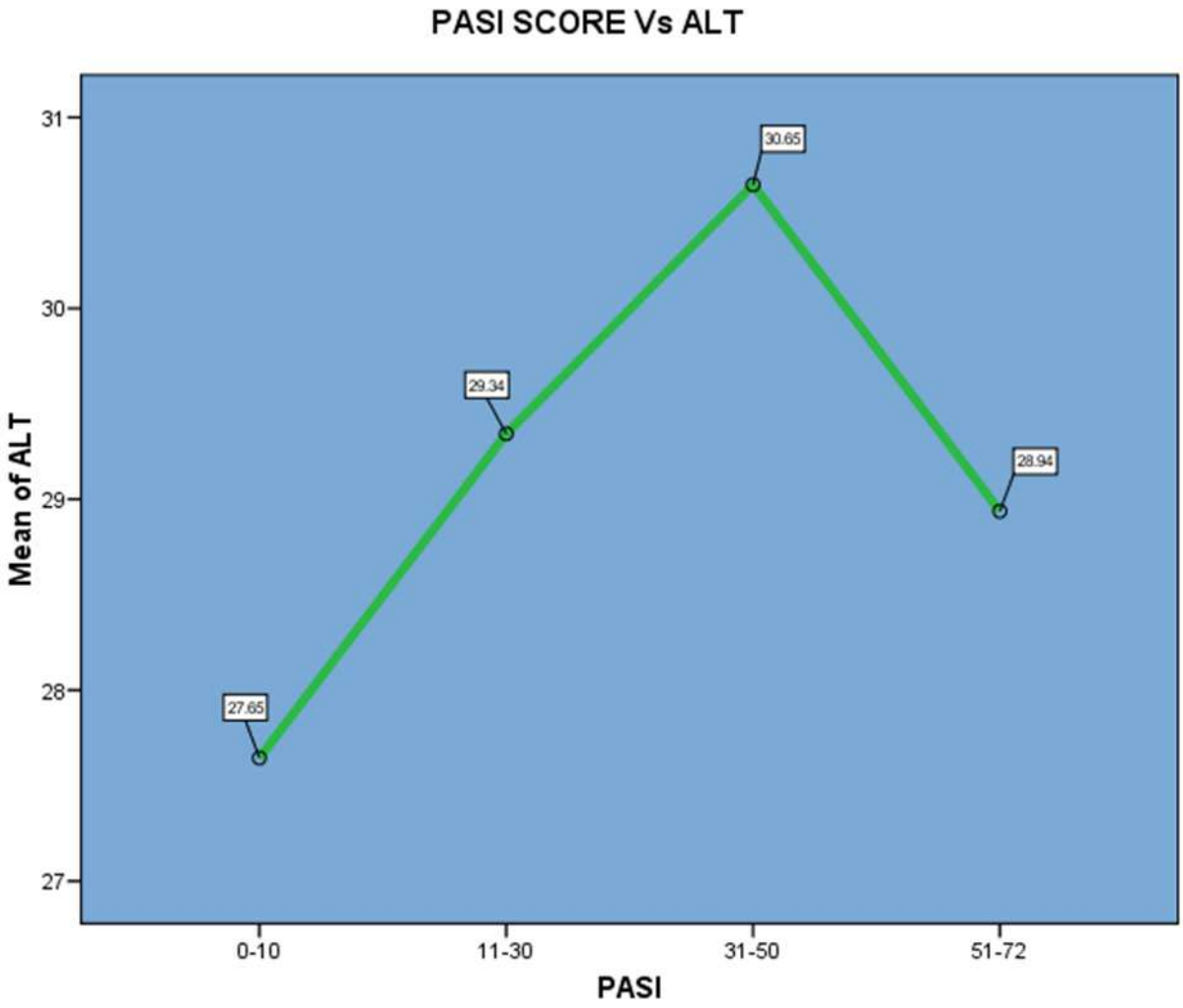
FIGURE 13


PASI SCORE Vs FREQUENCY OF FATTY LIVER
FREQUENCY OF FATTY LIVER
40
30
20
10
0
0-10
11-30
31-50
51-72
PASI
FATTY LIVER
NORMAL

AST LEVEL IN OUR STUDY



ALT LEVEL IN OUR STUDY



PSORIASIS AND METABOLIC SYNDROME

In our study 27 patients are found to have metabolic syndrome. Most of them are in the age group >40 and in PASI group 11-30 and predominately males.

17 of them are in PASI score of 11-30

16 patients had the duration of illness >3years

AGE AND SEX DISTRIBUTION OF METABOLIC SYNDROME

	AGE		**SEX**	
	<40	**>40**	**MALE**	**FEMALE**
NO.OF PATIENTS MS	7(4%)	20(12%)	16(9%)	11(6%)
TOTAL	27(16%)		27(16%)	

DISCUSSION

DISCUSSION

Psoriasis is a systemic illness involving almost all system in the body. It is a chronic inflammatory state , mediated by T helper cell and its cytokines. The role of adipokines in psoriasis is of recent interest. Various experiments have proved its role in psoriasis.

Of the comorbidities, metabolic syndrome is more prevalent among psoriasis patients and is considered as a high risk for developing coronary artery disease. NAFLD is now regarded as the hepatic manifestation of metabolic syndrome. Hence our objective of the study is to find the magnitude of NAFLD and metabolic syndrome among psoriasis patients.

The prevalence of NAFLD among normal population is estimated as 20-30% across various countries. The prevalence of NAFLD in India is reported as 9-19% in adult population.

PSORIASIS AND NAFLD:

In our study NAFLD is found in 75(45.5%) of the patients. Similar to previous other studies, the prevalence of NAFLD is increased in our study. Van der voort et al. study have documented 46.2% prevalence of NAFLD in psoriasis . Miele et al has reported a prevalence of 59.2%.among psoriasis patients. Gisondi et al, have found a prevalence of NAFLD in 47% of

patients with psoriasis. Madanagobalane et al showed a prevalence of 17.4% in south Indian population.

In our study NAFLD is seen distributed equally in all age group ,both the sex, and irrespective of the duration of illness. This is similar to the study by Gisondi et al in which the prevalence of NAFLD did not vary with age, gender, body mass index, psoriasis duration.

Compared to other studies like Gisondi et al, who had observed more prevalence of NAFLD in chronic plaque psoriasis and Madanagobalane et al study in which they found an association of NAFLD with psoriatic arthritis, we did not find any association with any specific type of psoriasis.

Our study showed predominant distribution of psoriasis patients with NAFLD in PASI group 11-30 years as compared with other studies like Gisondi et al study which also showed a correlation of NAFLD with the severity of psoriasis and higher PASI score.

Our psoriasis pateints with NAFLD are found to be associated with obesity ,dyslipidemia, and metabolic syndrome in our study. Other studies have reported similar observation. This is probably due to a similar pathogenesis which is common to both psoriasis and NAFLD. Adipocytokines have a role in psoriasis as well as NAFLD. Hypoadiponectinemia is associated with psoriasis. Takahashi et al have found

a correlation of decreased adiponectin levels with psoriasis disease severity. Shibata et al have shown that serum adiponectin levels increases after psoriatic therapy.

PSORIASIS AND METABOLIC SYNDROME

The prevalence of metabolic syndrome in our study is 27(16%).All other components are also increased in our study.

PREVALENCE OF VARIOUS COMPONENTS OF METABOLIC SYNDROME IN OUR STUDY.

TOTAL NO.OF PATIENTS-165

COMPONENTS OF MS	NO,OF PATIENTS
OBESITY	39(24%)
HT	43(26.1%)
FPS	55(33.3%)
TGL	60(36%)
HDL	65(39%)
CHOL	35(21%)
FATTY LIVER	75(45.5%)
MS	27(16%)

In our study metabolic syndrome is present predominately in males, above 40 years. The components of metabolic syndrome like hypertension, central obesity, diabetes are also seen predominately in above 40years. A study by Gisondi et al , and Madanagobalane et al also showed a similar result.

Madanagobalane et al study showed a prevalence of 44.1% of metabolic syndrome .Other studies like Safiye kutlu et al study ,Gisondi et al quote a prevalence of 30.8 % and 30.1% respectively.

Our psoriatic patients are found to have higher prevalence of central obesity 39(24%) and dyslipidemia which includes low HDL 65(39%), triglycerides 60(36%) and cholesterol 35(21%). This is similar to other study like Madanagobalane et al where they have observed an increased prevalence of triglyceridemia (33.9%) abdominal obesity (34.7%), but there was no difference in the high density lipoprotein (HDL) levels . Gisondi et al have observed no significant difference in prevalence on low HDL, Whereas Nazhatun nisa et al have observed a higher prevalence of low TGL. Other studies conducted in Iran ,UK, Hyderabad have found significant dyslipidemia in psoriasis.

Our study found a correlation of increased prevalence of metabolic syndrome in those with longer duration of illness, and in those with hyperglycemia. We did not find any correlation of metabolic syndrome with

the severity of psoriasis, hypertension , diabetes. Study by Madanagobalane et al showed no correlation with severity of psoriasis, duration , hypertension and HDL. Study by Neimann AL showed a higher prevalence of diabetes in severe psoriasis.

Almost all the patients with metabolic syndrome had NAFLD in our study, similar to other studies

CONCLUSION

CONCLUSION

Psoriasis is significantly associated with NAFLD and metabolic syndrome.

Increased prevalence of NAFLD indicates that psoriasis is independently associated with NAFLD.

Physicians should consider the possibility of chronic hepatic involvement in psoriasis before administering any hepatotoxic drug.

Psoriasis is no longer just skin deep, it is a chronic inflammatory multi system disease.

Patients should be screened for nonalcoholic fatty liver before administering any hepatotoxic drug and treatment planning should be done taking into consideration of various cardio metabolic comorbidities.

Psoriatic patients with metabolic syndrome should be educated about lifestyle modifications and they should be administered cardio protective drugs along with the psoriasis medications.

Psoriatic patients should be regularly screened for diabetes, atherosclerosis, and liver disease.

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BIBILIOGRAPHY

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ANNEXURES

ABBREVIATIONS

FFA	:	Free fatty acid
SAPHO	:	Synovitis, acne, Pustulosis, hyperostosis, and osteitis
PsA	:	Psoriatic arthritis
ALT	:	Alanine aminotransferase
ALP	:	Aspartate aminotransferase.
ANA	:	Anti-nuclear antibody
USG	:	Ultrasonogram
CT	:	Computed tomography
MRI	:	Magnetic Resonance Imaging
TGL	:	Triglycerides
LDL	:	Low density lipoprotein
CHOL	:	Cholesterol
HDL	:	High density lipoprotein
VLDL	:	Very low density lipoprotein
Th1	:	T helper cell 1

APC	:	Antigen presenting cell
TNF alpha	:	Tumor necrosis factor alpha
IL	:	Interleukin
VEGF	:	Vascular endothelial growth factor
CAD	:	Coronary artery disease
CVD	:	Cardiovascular disease
BMI	:	Body mass index
PASI	:	Psoriasis Area Severity Index
BSA	:	Body surface area
VAS	:	Visual analogue scale
NAFLD	:	Nonalcoholic fatty liver
COPD	:	Chronic obstructive pulmonary disease
DM	:	Diabetes mellitus
MS	:	Metabolic syndrome
HT	:	Hypertension
NCEP –ATP III	:	National cholesterol Education Programmes Adult Panel III

PROFORMA

Prevalence of nonalcoholic fatty liver disease and metabolic syndrome in psoriasis in a tertiary health center

Name : Patient ID No:

Age/Sex :

Duration of illness: Contact No:

TYPE OF PSORIASIS

☐Chronic plaque psoriasis ☐Psoriasis vulgaris

☐ Psoriatic arthritis ☐Palmoplantar psoriasis

☐Unstable psoriasis ☐Erythrodermic psoriasis

☐Pustular psoriasis

PASI SCORE

☐0-10 ☐10-30☐30-50 ☐50-72

EXAMINATION : BP: sitting mmHg;

WEIGHT: HEIGHT: BMI:

Waist circumference

INVESTIGATIONS

Fasting blood glucose: mg/dl

Fasting lipid profile:

Serum cholesterol mg/dl

Serum triglycerides mg/dl

Serum HDL mg/dl

Liver function test:

Serum bilirubin mg/dl

Aspartate aminotransferase(AST) IU/L

Alanine aminotransferase (ALT) IU/L

AST/ALT

CRP

Urine microalbumin: mg/dl

Ultrasonogram of abdomen:

Anti HCV antibody - HBs Ag-

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI-3

EC Reg No.ECR/270/Inst./TN/2013
Telephone No : 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. S. Abarna Devi,
Post Graduate, MD (General Medicine)
Institute of Internal Medicine,
Madras Medical College,
Chennai – 600003.

Dear Dr. S. Abarna Devi,

The Institutional Ethics Committee has considered your request and approved your study titled **"PREVALENCE OF NONALCOHOLIC FATTY LIVER DISEASE AND METABOLIC SYNDROME IN PSORIASIS IN A TERTIARY HEALTH CENTER"** No. 47072014.

The following members of Ethics Committee were present in the meeting held on 01.07.2014 conducted at Madras Medical College, Chennai-3.

1.	Dr. C. Rajendran, M.D.	-- Chairperson
2.	Dr. R. Vimala, M.D., Dean, MMC, Ch-3.	-- Deputy Chair Person
3.	Prof. Kalaiselvi, MD., Vice-Principal, MMC, Ch-3	-- Member Secretary
4.	Prof. Nandhini, M.D. Inst. of Pharmacology, MMC, Ch-3.	-- Member
5.	Dr. G. Muralidharan, Director Incharge , Inst. of Surgery	-- Member
6.	Prof. Md Ali, MD., DM., Prof & HOD of MGE, MMC, Ch-3.	-- Member
7.	Prof. Ramadevi, Director i/c, Biochemistry, MMC,Ch-3.	-- Member
8.	Prof. Saraswathy, MD., Director, Pathology, MMC, Ch-3.	-- Member
9.	Prof. Tito, Director, i/c. Inst. of Internal Medicine, MMC	-- Member
10.	Thiru. Rameshkumar, Administrative Officer	-- Lay Person
11.	Thiru. S. Govindasamy, BABL, High Court, Chennai-1.	-- Lawyer
12.	Tmt. Arnold Saulina, MA MSW	-- Social Scientist

We approve the proposal to be conducted in its presented form.

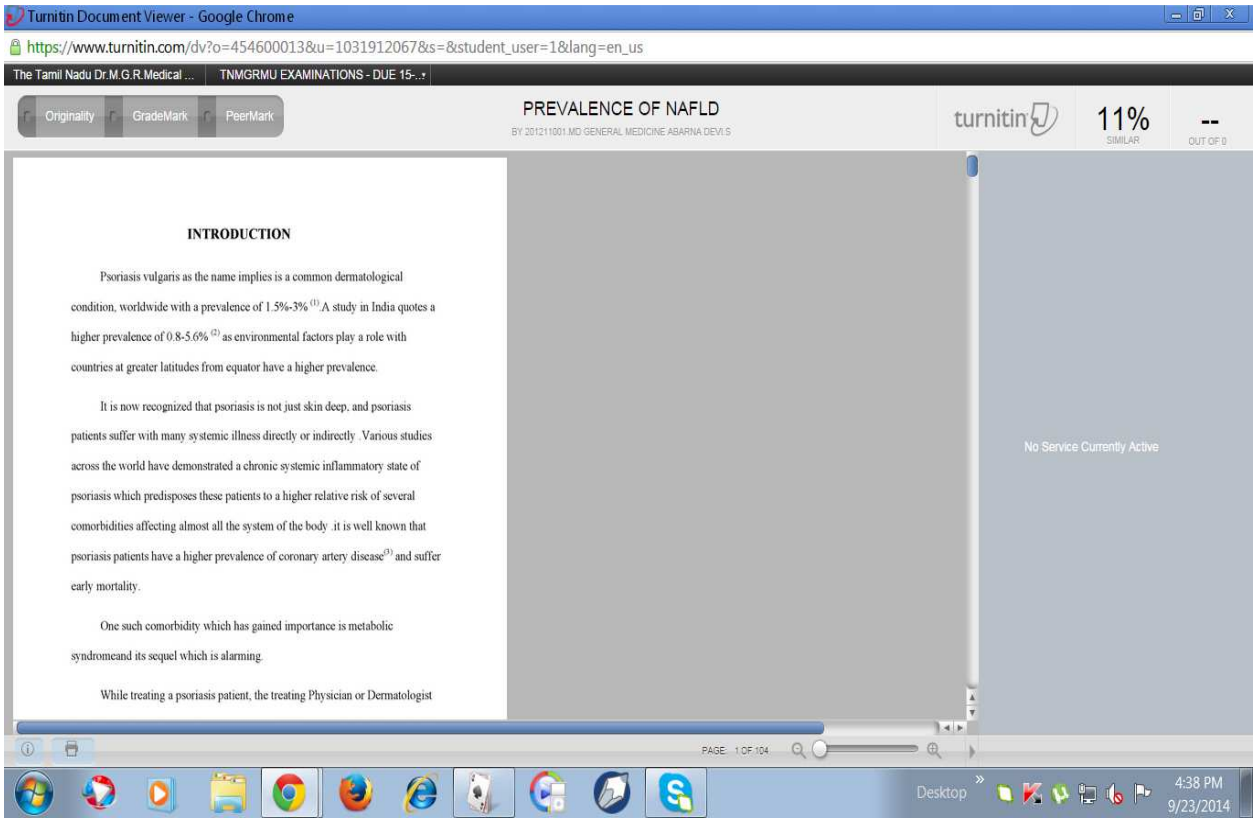
Sd/Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

TURNITIN PLAGIARISM SCREEN SHOT



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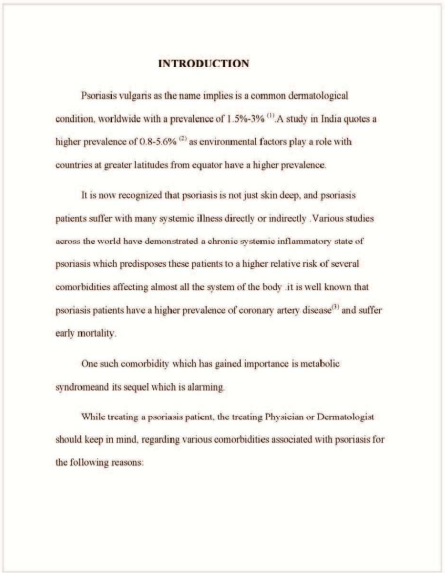

INTRODUCTION

Psoriasis vulgaris as the name implies is a common dermatological condition, worldwide with a prevalence of 1.5%-3% [1].A study in India quotes a higher prevalence of 0.8-5.6% [2] as environmental factors play a role with countries at greater latitudes from equator have a higher prevalence.

It is now recognized that psoriasis is not just skin deep, and psoriasis patients suffer with many systemic illness directly or indirectly .Various studies across the world have demonstrated a chronic systemic inflammatory state of psoriasis which predisposes these patients to a higher relative risk of several comorbidities affecting almost all the system of the body .it is well known that psoriasis patients have a higher prevalence of coronary artery disease[3] and suffer early mortality.

One such comorbidity which has gained importance is metabolic syndromeand its sequel which is alarming.

While treating a psoriasis patient, the treating Physician or Dermatologist should keep in mind, regarding various comorbidities associated with psoriasis for the following reasons:

ஆராய்ச்சி தகவல் தாள்

சென்னை அரசு பொது மருத்துவமனைக்கு வரும் சொரியாஸிஸ் நோயாளிகளுக்கு ஏற்படும் (NAFLD) கல்லீரல் நோய் மற்றும் ''மெடபாலிக் சின்ட்ரோம்'' பற்றிய ஒரு ஆராய்ச்சி இங்கு நடைபெற்று வருகின்றது.

சொரியாஸிஸ் நோயாளிகளின் இடுப்பளவு, எடை மற்றும் இரத்த அழுத்தம் முதலில் கண்டறியப்படும். பிறகு காலை வெறும் வயிற்றில் 5 மி.லி. இரத்தம் எடுக்கப்பட்டு அதில் சர்க்கரை, கொழுப்பு C-reactive Protein மற்றும் கல்லீரல் குறைபாடுகள் கண்டறியப்படும். சிறுநீரில் மைக்ரோ அல்புமின் பார்க்கப்படும். இதே நோயாளிகளுக்கு வயிறு ஸ்கேன் செய்யப்படும். இதில் கல்லீரல் வீக்கம் (Fatty Liver) கண்டறியப்பட்டால் ஃபைப்ரோஸ்கேன் செய்யப்படும்.

அதனால் தங்களது நோயின் ஆய்வறிக்கையோ அல்லது சிகிச்சையோ பாதிப்பு ஏற்படாது என்பதையும் தெரிவித்துக்கொள்கிறோம்.

முடிவுகளை அல்லது கருத்துக்களை வெளியிடும்போதோ அல்லது ஆராய்ச்சியின்போதோ தங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிட மாட்டோம் என்பதை தெரிவித்துக்கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின்பேரில்தான் இருக்கிறது. மேலும் நீங்கள் எந்த நேரமும் இந்த ஆராய்ச்சியிலிருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

இந்த சிறப்பு பரிசோதனைகளின் முடிவுகளையும் நோயின் தன்மை பற்றியும் ஆராய்ச்சியின்போது அல்லது ஆராய்ச்சியின் முடிவின்போது தங்களுக்கு அறிவிப்போம் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

ஆராய்ச்சியாளர் கையொப்பம் **பங்கேற்பாளர் கையொப்பம்**

நாள் :
இடம் :

INFORMATION SHEET

We are conducting a study on **"Prevalence of nonalcoholic fatty liver disease and metabolic syndrome in psoriasis in a tertiary health center"** among patients attending Rajiv Gandhi Government General Hospital, Chennai and for that your specimen may be valuable to us.

The purpose of this study is to assess prevalence of non alcoholic fatty liver disease and metabolic syndrome in psoriasis. We are selecting certain cases and if you are found eligible, blood pressure, BMI, waist circumference will be checked. 5ml blood will be collected in fasting state and 2ml of urine will be collected. You will also undergo ultrasound abdomen and if found to have liver enlargement fibro scan will be taken. These tests and special studies do not affect your final report or management.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of Investigator	Signature of Participant

Date :

Place :

ஆராய்ச்சி ஒப்புதல் கடிதம்

ஆராய்ச்சி தலைப்பு
சொறியாஸிஸ் நோயாளிகளுக்கு ஏற்படும் (NAFLD) கல்லீரல் நோய் மற்றும் "மெடபாலிக் சின்ட்ரோம்" பற்றிய ஒரு ஆராய்ச்சி

பெயர்	:	தேதி	:
வயது	:	உள் நோயாளி எண்	:
பால்	:	ஆராய்ச்சி சேர்க்கை எண்	:

இந்த ஆராய்ச்சின் விவரங்களும் அதன் நோக்கங்களும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது.

எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்துகொண்டு எனது சம்மதத்தை தெரிவிக்கிறேன்.

இந்த ஆராய்ச்சியில் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின்பேரில் பங்கு பெறுகின்றேன். இந்த ஆராய்ச்சியில் இருந்து நான் எந்நேரமும் பின்வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்துகொண்டேன்.

எனக்கு கல்லீரல் நோய் சம்பந்தப்பட்ட ரத்த பரிசோதனைகளும், வயிறு ஸ்கேன், ஃபைப்ரோ ஸ்கேன் செய்துகொள்ள சம்மதிக்கிறேன்.

நான் சொறியாஸிஸ் நோயில் ஏற்படும் கல்லீரல் பாதிப்பு மற்றும் மெடபாலிக் சிண்ட்ரோம் குறித்த இந்த ஆராய்ச்சியின் விபரங்களைக் கொண்ட தகவல் தாளைப் பெற்றுக்கொண்டேன்.

நான் என்னுடைய சுய நினைவுடனும் மற்றும் முழு சுதந்திரத்துடனும் இந்த மருத்துவ ஆராய்ச்சியில் என்னை சேர்த்துக்கொள்ள சம்மதம் தெரிவிக்கிறேன்.

கையொப்பம்

PATIENT CONSENT FORM

Study Title : **Prevalence of nonalcoholic fatty liver disease and metabolic syndrome in psoriasis in a tertiary health center**

Study Centre : Rajiv Gandhi Government General Hospital, Chennai.

Name :

Age/Sex :

Identification Number :

Patient may check (☑) these boxes

The details of the study have been provided to me in writing and explained to me in my own language ❑

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ❑

I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ❑

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or wellbeing or any unexpected or unusual symptoms. ❑

I hereby consent to participate in this study. ❑

I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological and biochemical tests. ❑

Signature/thumb impression

Patient's Name and Address:

Signature of Investigator

Study Investigator's Name:

Dr. S.ABARNA DEVI

MASTER CHART

S.no	AGE	SEX	BMI	W-C	BP	FBS	TGL	CHOLES	HDL	AST	ALT	USG	F SCAN	PASI	TYPE	DURATION
1	25	Male	26.7	89	120/70	97	183	232	41	22	27	FL	7.1	10	CPP	1 YR
2	46	Male	28.9	109	110/80	88	244	188	29	34	44	FL	10.1	10	CPP	2
3	53	Male	27	103	130/90	101	570	282	30	102	40	FL	4.7	40	PA	2
4	39	Male	28	98	120/80	150	157	300	34	19	15	N		10	CPP	3
5	42	Female	36	105	120/80	87	379	181	40	25	36	FL	4.7	30	CPP	3
6	54	Male	40	106	120/80	96	124	191	44	17	28	FL	6.8	10	PA	1
7	27	Male	22	81	120/80	87	109	209	54	18	11	N		20	CPP	1
8	75	Male	28	99	150/80	160	160	205	34	23	20	FL	3.8	20	PP	5
9	70	Male	29	85	130/70	78	167	174	53	24	30	FL	3.8	10	NP	4
10	42	Male	25.48	90	120/80	95	139	196	38	23	13	FL	4	30	PA	3
11	33	Female	27.35	94	90/70	90	32	160	48	28	34	FL	4.5	20	CPP	4
12	63	Male	25	92	160/80	98	49	150	45	26	19	FL	4.5	10	CPP	5
13	45	Male	22.83	86	120/80	98	80	140	36	30	30	FL	4.6	10	CPP	3
14	55	Male	24.15	90	120/80	105	304	175	41	20	19	FL	4.9	20	CPP	6
15	44	Female	32.43	89	130/90	180	305	214	42	21	27	FL	4.7	30	PPP	6
16	43	Male	25	96	120/80	103	292	252	40	31	40	FL	9.7	20	PPP	4
17	45	Male	24.11	90	110/80	67	255	239	54	34	42	FL	6.7	20	PPP	2
18	50	Male	25.39	43	130/100	116	138	174	48	18	18	FL	5.5	20	PP	2
19	62	Male	24	85	120/80	88	90	142	43	24	11	N		10	PPP	2
20	57	Male	20.44	84	120/80	110	180	10	35	21	20	FL	3.8	20	CPP	1
21	20	Female	24	86	110/80	101	130	130	56	26	16	FL	3.8	10	PUP	5
22	42	Male	30.4	107	110/80	89	130	130	46	24	20	FL	4.7	30	CPP	6
23	57	Male	25.61	87	120/80	78	120	110	38	22	30	FL	3.8	10	CPP	4
24	63	Female	25	87	140/80	99	110	102	36	24	30	FL	4.7	15	CPP	2
25	60	Male	25	100	110/80	98	229	303	55	35	35	FL	4.6	25	CPP	3
26	38	Male	23	76	140/90	110	338	313	61	26	39	FL	4.7	10	SP	5
27	48	Male	19	74	110/80	99	165	110	44	34	30	N		20	CPP	6

28	52	Male	28.8	115	120/80	88	138	175	48	28	3	N		10	SP	3
29	40	Male	25.4	62	130/80	90	206	250	35	22	16	FL	4.6	22	PUP	2
30	60	Male	23	58	130/80	99	180	230	45	28	52	FL	5	24	CPP	2
31	45	Female	30	106	150/80	115	170	110	54	22	23	N		30	PA	5
32	62	Male	27	96	120/80	85	40	120	65	22	10	FL	4.6	40	PPP	3
33	28	Female	28	95	100/60	79	104	166	43	15	10	N		20	PPP	2
34	48	Male	26	85	130/90	98	74	149	34	25	20	N		20	CPP	4
35	60	Male	25	84	120/80	102	150	130	36	32	20	FL	4.6	25	CPP	2
36	65	Male	30	109	180/100	110	168	196	54	24	20	FL	5.6	25	CPP	3
37	72	Female	31	122	170/80	160	160	150	45	23	30	FL	4.6	45	CPP	2
38	52	Male	24	73	130/80	110	150	120	35	30	30	N		35	CPP	2
39	28	Male	23	69	120/80	106	140	110	36	30	40	N		44	CPP	1
40	18	Male	22	69	130/90	89	130	90	36	28	20	FL	4.6	35	UP	1
41	72	Male	22	60	120/80	87	120	69	33	20	32	N		20	CPP	1
42	43	Male	21	59	110/80	78	130	89	45	27	40	FL	4.9	10	CPP	1
43	52	Male	20	74	105/93	98	160	57	35	39	33	N		12	NP	2
44	28	Male	19	72	120/80	99	150	98	56	30	20	N		10	PPP	2
45	54	Female	18	86	120/80	87	150	110	35	32	20	N		10	PPP	3
46	34	Male	16	74	120/80	79	140	166	25	20	20	N		13	PPP	4
47	26	Male	17	45	110/80	98	98	152	54	22	24	N		60	PE	2
48	43	Male	21	48	110/80	99	150	150	69	21	37	FL	3.7	20	CPP	1
49	28	Female	23	90	110/80	80	160	140	40	34	28	FL	4.8	25	CPP	1
50	38	Female	22	76	110/80	90	160	130	56	23	25	FL	4.7	20	CPP	2
51	30	Female	26.22	59	110/70	93	150	120	34	58	39	N		20	CPP	2
52	34	Female	26	60	120/80	98	166	110	70	48	20	N		20	CPP	2.2
53	27	Female	14	75	120/80	97	156	90	43	44	26	N		20	CPP	2.6
54	48	Male	28	84	110/80	97	164	67	33	23	14	N		10	CPP	2.4
55	23	Male	21	68	130/80	96	165	136	32	40	30	FL	4.6	10	CPP	2
56	65	Male	24	66	130/86	88	180	110	20	21	11	N		10	CPP	2
57	71	Female	21	65	110/80	87	163	205	55	50	40	N		10	CPP	3
58	61	Male	32	106	110/80	88	164	110	30	46	30	N		15	PPP	4

59	25	Male	17	87	130/90	86	149	105	55	22	34	N		20	PPP	4
60	53	Male	21.8	76	130/90	85	80	203	38	59	20	N		10	PA	5
61	54	Female	30	100	110/80	87	153	108	36	20	29	N		10	PA	3
62	45	Male	27.7	58	120/80	88	227	221	63	40	20	FL	7.7	20	PPP	2
63	42	Male	30	109	110/80	99	143	108	46	44	30	N		10	PPP	2
64	43	Male	24	68	130/80	90	190	160	36	30	40	FL	5.8	10	CPP	2
65	21	Male	22	54	90/60	98	146	160	55	50	30	N		16	CPP	2
66	51	Male	24	45	142/80	90	130	226	20	26	30	FL	6.3	30	CPP	2
67	32	Female	31	110	178/120	87	203	155	33	25	28	N		20	CPP	3
68	62	Male	25	55	100/70	65	113	205	37	40	30	N		20	CPP	1
69	28	Male	21	57	90/80	87	134	159	45	30	30	N		10	CPP	1
70	21	Female	25.67	68	120/88	100	130	120	33	18	15	FL	4.8	10	CPP	1
71	62	Female	30	98	110/70	112	138	150	40	20	30	N		10	GP	1
72	26	Female	28	88	110/80	152	154	150	24	29	22	N		20	GP	1
73	47	Female	25	46	110/80	124	130	140	50	30	40	N		10	PPP	1
74	58	Male	26	57	120/80	120	120	130	30	30	20	N		18	CPP	2
75	61	Female	27	56	110/80	87	110	140	50	30	20	N		13	CPP	2
76	70	Female	28	45	130/90	89	90	140	40	35	30	N		15	CPP	2
77	50	Male	25.74	47	130/70	90	218	271	51	30	20	FL	4.5	15	CPP	3
78	30	Male	25	60	120/90	90	148	150	30	39	30	FL	5.8	25	CPP	3
79	40	Male	24	56	120/80	98	130	148	44	20	12	N		14	CPP	4
80	43	Female	26.6	78	110/80	90	200	130	50	45	20	FL	4.4	60	PP	5
81	42	Female	19	88	110/80	89	120	120	30	35	30	FL	4.7	45	UP	1.5
82	61	Male	18	87	110/80	89	110	110	40	28	34	FL	4.7	10	SP	2.6
83	43	Female	19	47	110/80	87	67	108	44	30	33	FL	4.7	10	SP	1.6
84	34	Female	18	45	110/80	98	89	110	30	40	25	N		10	SP	1.7
85	50	Female	28	69	110/80	88	66	120	35	40	44	N		20	CPP	0.7
86	42	Female	27	68	130/70	89	88	130	39	17	35	N		26	CPP	0.4
87	28	Male	26	66	120/90	87	83	110	37	30	35	N		30	CPP	0.7
88	38	Male	24	65	130/90	76	78	120	34	30	45	FL	5.7	10	PA	0.3
89	24	Male	19	55	130/90	99	66	109	66	33	33	FL	5.8	10	PA	0.8

90	28	Male	18	45	120/90	98	89	108	40	40	33	FL	5.7	10	PA	0.6
91	19	Male	15	38	110/80	87	99	150	40	30	29	FL	6.3	30	CPP	3.6
92	22	Female	15	45	110/80	200	89	120	46	30	24	N		20	CPP	0.8
93	36	Female	18	45	120/80	87	76	160	20	44	33	FL	6.5	10	CPP	6
94	41	Female	17	45	120/80	110	110	120	50	50	33	N		10	PA	4.6
95	55	Male	21	45	100/80	195	134	159	31	17	22	N		24	CPP	4
96	54	Male	30	56	110/80	90	120	205	55	40	54	FL	4.8	20	CPP	3
97	35	Female	28	56	110/80	98	128	262	51	25	44	N		10	PPP	2.5
98	54	Female	26.66	55	105/80	191	209	120	40	25	28	FL	4.4	20	CPP	2
99	72	Male	24	50	120/70	66	35	235	36	14	18	N		10	PPP	1
100	32	Female	19	46	120/80	117	380	150	43	20	24	N		10	PPP	3
101	57	Male	34.37	120	150/80	87	110	140	33	30	26	FL		10	CPP	1.2
102	64	Male	25	63	130/80	99	255	120	40	30	22	FL	3.9	10	CPP	2
103	43	Male	19.33	55	110/80	97	110	130	47	40	24	N		10	CPP	2
104	54	Male	32	125	130/90	96	111	120	30	36	32	FL		10	CPP	3
105	55	Male	30	111	130/90	96	67	120	32	49	24	FL		20	PA	1
106	26	Male	23	50	120/80	180	89	180	50	48	55	N		20	PP	1.1
107	27	Female	24	55	120/80	98	90	150	40	48	38	N		10	SP	1.2
108	28	Male	20	51	110/70	99	87	140	30	38	39	N		10	SP	1.5
109	29	Female	25	48	130/90	90	67	108	44	28	38	N		10	PA	1.4
110	55	Male	24	96	130/80	96	89	219	35	40	39	N		20	PA	1.5
111	45	Male	20	73	90/60	96	165	184	44	40	33	FL		25	PV	0.5
112	39	Male	21	88	110/70	137	112	120	54	30	33	FL		60	UP	0.6
113	65	Female	23.45	98	120/80	73	90	110	33	44	28	N		20	PV	0.7
114	26	Male	19.32	58	140/80	68	90	108	40	33	23	N		15	PV	4
115	72	Male	23.27	66	113/80	175	98	130	40	21	14	N		15	PV	10
116	27	Male	21.75	56	110/70	104	141	222	51	21	30	FL		30	PV	2.5
117	52	Female	25.86	66	140/80	105	322	202	45	17	17	FL	4.8	20	PA	0.6
118	27	Female	22	56	120/80	110	121	234	57	27	20	FL	4.8	10	PV	1
119	29	Male	24.85	68	110/80	106	300	261	46	24	29	N		10	PV	4
120	42	Male	25.95	78	101/88	99	157	165	58	48	10	N		60	PV	3.5

121	36	Female	22.31	58	110/80	96	67	140	48	27	53	N		30	PV	2
122	36	Male	24.16	56	110/70	87	191	267	33	38	10	FL	5.9	10	PV	9
123	42	Male	21.13	45	110/90	140	156	259	48	12	13	FL	5.8	14	CPP	0.6
124	42	Male	30.17	112	140/90	109	67	150	52	32	39	FL	4.6	15	CPP	3
125	36	Female	30	98	130/90	182	345	237	55	13	10	FL	8.3	15	PA	5
126	35	Female	22.5	46	104/80	108	89	130	42	22	12	N		20	UP	8
127	30	Female	33.5	98	120/80	98	88	120	36	26	33	N		10	SP	10
128	8	Male	21.6	56	130/80	96	88	130	79	30	45	N		10	NP	0.5
129	65	Male	25.6	46	100/80	98	76	120	30	30	37	N		10	GP	0.5
130	30	Male	27.89	76	120/80	98	66	110	44	30	33	N		20	GP	0.7
131	35	Male	18	45	130/80	91	143	237	45	30	32	N		10	SP	3
132	30	Female	24.5	65	130/80	125	88	130	30	30	28	FL	5	10	PPP	0.2
133	58	Female	26	56	109/70	325	75	120	57	19	29	N		10	PPP	0.5
134	45	Female	21	43	113/77	109	77	130	23	22	22	N		10	SP	1.5
135	65	Male	28	45	120/80	126	78	120	40	22	40	FL	4.8	10	PPP	0.5
136	18	Male	26.6	76	110/70	90	161	218	58	20	8	FL	4.5	10	PPP	0.6
137	48	Male	16	45	120/80	117	55	130	58	28	26	N		20	CPP	0.6
138	40	Female	26.6	47	110/80	104	46	120	44	42	58	N		20	CPP	0.8
139	54	Female	27.16	76	120/80	112	164	219	52	20	12	N		20	UP	3
140	39	Female	22	64	150/90	105	287	232	44	25	25	N		10	SP	1.3
141	37	Female	26	33	108/75	136	220	195	41	23	18	FL		10	PA	0.1
142	46	Female	26	45	102/80	102	116	198	44	19	16	N		10	NP	0.2
143	32	Male	15	37	108/60	96	124	191	34	11	19	FL	5.3	10	NP	0.4
144	58	Male	27	46	120/80	89	55	150	38	76	56	FL	4.8	10	SP	0.6
145	45	Male	23	43	120/80	97	55	140	49	40	30	FL	4.6	10	PA	1
146	29	Female	21.6	34	110/80	120	56	130	34	20	44	N		15	CPP	0.1
147	34	Male	30	98	110/80	124	191	158	68	30	33	N		15	CPP	0.3
148	27	Female	31	97	120/80	124	78	160	44	39	34	N		15	CPP	0.6
149	54	Female	32	90	130/90	122	88	140	46	20	46	FL	4.9	15	CPP	3
150	50	Male	18	78	120/80	120	88	130	57	40	32	N		14	CPP	1
151	22	Male	32	88	130/90	110	78	150	46	39	54	N		36	CPP	11

152	34	Male	28	78	130/90	110	76	140	34	19	36	N		24	PA	2
153	32	Female	22	48	120/80	90	55	150	67	18	28	N		20	PA	1
154	27	Female	26	39	110/80	99	127	133	45	18	49	N		66	PE	1
155	54	Male	24	48	120/80	140	78	140	45	20	36	N		40	PV	1
156	32	Female	25	59	130/80	89	99	130	47	20	44	N		20	PV	3
157	12	Female	29	56	120/80	89	108	216	36	20	28	N		10	PV	5
158	14	Female	19	43	110/80	87	78	150	68	25	37	N		10	PV	3
159	16	Female	16	37	110/86	77	103	158	48	49	30	N		10	PV	2
160	18	Female	18	56	120/80	90	110	140	30	20	30	FL	4.4	10	PV	0.5
161	31	Female	18	45	120/80	98	110	120	35	12	32	N		15	PV	0.5
162	20	Female	19	35	120/80	90	84	237	51	20	38	N		16	PV	0.6
163	23	Female	22	56	110/80	97	88	130	36	40	20	N		20	PV	0.7
164	24	Female	23	54	110/80	110	68	120	60	20	30	FL	4.4	20	PV	2
165	46	Female	24.5	76	110/80	104	88	120	54	20	50	N		20	PV	2

KEY TO MASTER CHART

S.NO	-	Serial number
M	-	Male
F	-	Female
BMI	-	Body mass index
WC	-	Waist circumference in cm
BP	-	Blood pressure
FBS	-	Fasting blood sugar in mg/dl
FLP	-	Fasting lipid profile
TGL	-	Serum triglycerides in mg/dl
HDL	-	Serum high density lipoprotein in mg/dl
CHOL	-	Serum cholesterol in mg/dl
AST	-	Aspartate aminotransferase.
ALT	-	Alanine aminotransferase
USG	-	Ultrasonogram of abdomen
FL	-	Fatty liver
N	-	Normal
PASI	-	Psoriasis area severity index

DT	-	Duration of illness
PV	-	Psoriasis vulgaris
CPP	-	Chronic plaque psoriasis
PA	-	Psoriatic arthritis
PPP	-	Palmoplantar psoriasis
PUP	-	Pustular psoriasis
UP	-	Unstable psoriasis
SP	-	Scalp psoriasis
NP	-	Nail psoriasis
PE	-	Erythrodermic Psoriasis
GP	-	Guttate psoriasis
DT	-	Duration of illness
Y	-	Year